YEARLY SUBSCRIPTION, $6.00 SINGLE NUMBERS, $1.00

MAY 31 1923 Medical

# THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

[FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY]

OFFICIAL ORGAN OF

THE AMERICAN ROENTGEN RAY SOCIETY

AND THE AMERICAN RADIUM SOCIETY

*Editor:* H. M. IMBODEN, M.D.

*Associate Editors:*

JAMES T. CASE, M.D. H. K. PANCOAST, M.D.

EDITORIAL BOARD

FREDERICK H. BAETJER, M.D.
A. BECLERE, M.D.
DAVID R. BOWEN, M.D.
PERCY BROWN, M.D.
R. D. CARMAN, M.D.
L. G. COLE, M.D.
A. W. CRANE, M.D.
A. W. GEORGE, M.D.
PRESTON M. HICKEY, M.D.
ROBERT KNOX, M.D.
R. LEDOUX-LEBARD, M.D.
G. E. PFAHLER, M.D.
A. H. PIRIE, M.D.
E. H. SKINNER, M.D.

VOLUME X NUMBER 5

May, 1923

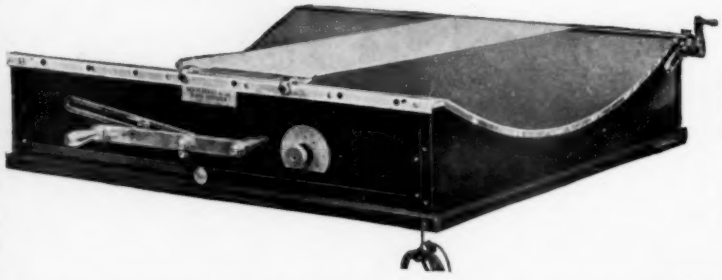
PUBLISHED MONTHLY BY PAUL B. HOEBER

NEW YORK CITY

TWENTY-FOURTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY WILL BE HELD AT CHICAGO, ILL., SEPTEMBER 18, 19, 20, 21, 1923.
EIGHTH ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY WILL BE HELD AT SAN FRANCISCO, CALIF., JUNE 25, 26, 1923.

Entered as second-class matter, March 3, 1916, at the Post Office, New York, N. Y., under the Act of March 3, 1879.

# BRADY'S POTTER-BUCKY DIAPHRAGM



## NEW FEATURES

A new and improved type of grid construction that secures even more brilliant results than our previous grid.

A neat compression band attachment for holding the patient still during exposure.

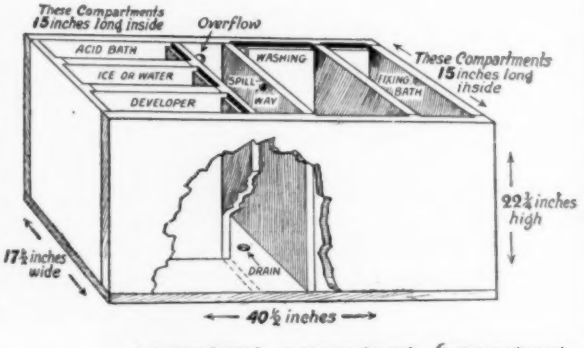
Patient brought nearer to plate. An electrical contact device for operating a signal lamp or which can be connected to most time switches for making the exposure automatically, from 1 to 90 seconds time at $5.00 extra charge.

Our speed control is accurate and reliable and timed in actual seconds. It does not require constant repairs. We claim it is the smallest, neatest and most effective Bucky on the market, 6:1 ratio grid cutting off practically 90% of secondary. Takes all makes of cassettes to 17 x 17 size, operates in horizontal or vertical position, and does Stereoscopic work in either direction.

All-metal construction electrically welded and practically indestructible. They do not get "out of date." Price $250.00 list with cash discount for quick payment.

## DEVELOPING TANKS

Our stone tanks are considered the best designs and values on the market. Each has a space for ice or hot water for controlling the developing temperature. The 5 and 6 compartment styles also have a 4" air space to prevent temperature of wash water from affecting the temperature of developer. Lengths over all 24½"-34"-40¼" respectively. We have enamel tanks also. Send for special circular.




| | 4-compartment | 5-compartment | 6-compartment |
|---|---|---|---|
| Prices shipped from Virginia : : : : | $45.00 | $53.50 | $60.00 |
| Prices shipped from Chicago, Boston or Brooklyn : | $50.00 | $58.50 | $65.00 |

## NEW INTENSIFYING SCREEN PRICES—GET OUR NEW NET PRICES

on standard makes of screens. Alone or mounted into our own or Buck Cassettes at prices that are sure to interest you. Will mount in your own make cassettes if preferred.

We allow very liberal discounts for prompt payment on screens and cassettes.

## BARIFLUFF—Our New Barium—*Light weight or "fluffy" type.*

For those who want a lighter Barium than our regular grade that has been the standard for nine years. The price is higher—but it is pure Barium containing no phosphates or other adulteration to make it float. You cannot afford to use anything but Pure barium. Ours analyzes over 99½%. Prices per 100 lb. $26.00—50 lb. $14.00—25 lb. $8.50.

## NEW DENTAL MOUNTS—*Ask for sample and prices.*

## NEW PRICE LIST *Now Ready*— SEND FOR A COPY—IT MAY SAVE YOU MONEY

*We allow liberal cash discounts on all supplies for quick payment.*

GEO. W. BRADY & CO., 761 S. Western Ave., Chicago

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. X MAY, 1923 No. 5

# CARDIAC PNEUMOFIBROSIS*

BY GEORGE W. HOLMES, M.D., AND DAVID S. DANN, M.D.

*X*-Ray Laboratory, Massachusetts General Hospital

BOSTON, MASSACHUSETTS

## INTRODUCTION

ALTHOUGH there has been a definite advance in the accuracy of interpretation of the radiographic findings in diseases of the chest during the past few years, the shadows appearing at the hilus and along the course of the bronchial tree are still a source of considerable confusion. It was in an attempt to explain some of these doubtful shadows that this study was undertaken.

We shall not discuss all of the various pathological conditions which may produce an increase in hilus and peribronchial density, but will mention only those which are known to produce fibrosis, and discuss more completely the pathology of the lungs and the radiographic appearance produced in chronic pulmonary congestion.

While it is generally known that the clinical picture in cardiac pneumofibrosis, particularly when it is the result of valvular disease of the heart, may simulate closely that of tuberculosis of the lungs even to the extent of hemorrhage, we have been able to find very little reference to the similarity of the radiographic appearances.

## REVIEW OF LITERATURE

Barjon[1] devotes a chapter to the changes in the lung due to vascular processes. He divides them into primary active congestion, secondary active congestion, passive congestion, edema and infarcts. Most of the text is given to a description of the acute process which gives a diffuse radioscopic shadow seen at the bases of the lungs somewhat resembling pneumonia. No illustrations are given.

Overend[2] reports a case with a radiograph showing the characteristic mitral configuration of the heart and increased density in the hilus and perihilus region. In discussing this case, he states that both pulmonary fields are pervaded by the fine mottling of diffuse fibrosis.

The following description (accompanied by two characteristic radiograms) from Assmann[3] is the best we have found in the literature:

"Stasis of blood in the capillaries and smaller veins of the lungs causes certain changes in the roentgenogram. These changes are seen in decompensation, particularly in mitral regurgitation, and also in myocarditis and other conditions affecting the left ventricle. The film in these cases has a sort of 'washed-out' appearance which gives the impression at first glance of its being badly exposed or faulty. The most marked case of this disturbance of the lung picture which I have seen is one of mitral stenosis in which there had been repeated attacks of severe hemoptysis. Another sign of pulmonary congestion which may not run exactly parallel to the amount of clouding of the lung fields is a broadening and increased prominence of the normal lung markings which are made up of the larger and medium-sized blood-vessels. Accompanying this there is also

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922.

increased prominence of the smaller markings lying adjacent.

"These increased markings run from the hilus toward the periphery, and they decrease uniformly in size and intensity as they go outward, for we are dealing with a condition which affects the blood-vessel system as a whole. In this respect this condition differs from the discrete shadows which are due to increase in the size of the lymphatic glands or local thickenings of the bronchial wall as seen in bronchiectasis.

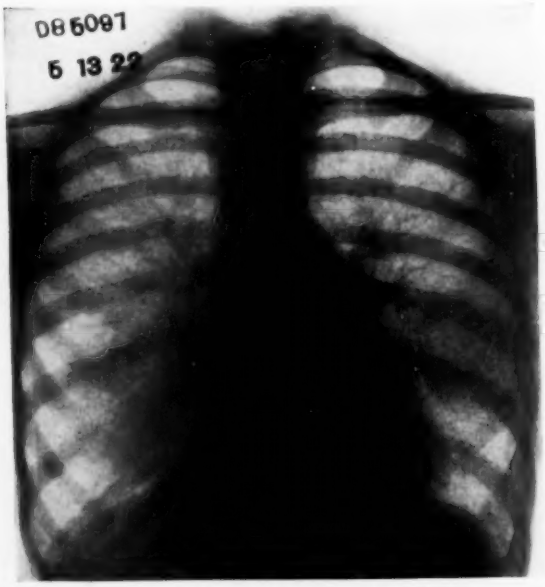



FIG. 1. Rheumatic heart disease with mitral stenosis in a child fourteen years of age. This plate shows the "washed-out" appearance characteristic of cardiac pneumofibrosis, marked increase in the hilus shadow and a general thickening and mottling of the larger lung markings (with the typical mitral shape of the cardiac shadow), making the picture a very typical one.

"This increase in the hilus shadows and of the blood-vessel markings radiating out from the hila in cases of pulmonary congestion has given the erroneous impression of its being peribronchial or lymphatic tuberculosis."

Stillman,[4] in discussing the clinical manifestations, states that when the heart is compensating, it produces no symptoms, but with failure of the cardiac muscle there is dyspnea, cyanosis and cough. The dyspnea is ascribed to diminished elasticity of the lung and the narrowing of the alveoli. The cyanosis usually parallels the cough and dyspnea intensity. The cough may be very annoying and bring up a moderate amount of sputum. Blood is quite commonly present, usually as streaks or small masses. The physical signs are not at all clear cut. There may be dullness, and moist rales at the bases, or, at times, no deviation from the normal can be made out.

Robinson,[5] in a description of mitral disease, states that the early and most characteristic effects result from disturbances of the pulmonary circulation. Paroxysmal attacks resembling asthma are sometimes seen. Cough, with expectoration sometimes bloody or blood-streaked, accrues. Hemoptysis is not uncommon. Bronchitis is a frequent complication. The signs of this condition and other pulmonary signs lead to confusion at times between mitral stenosis and tuberculosis.

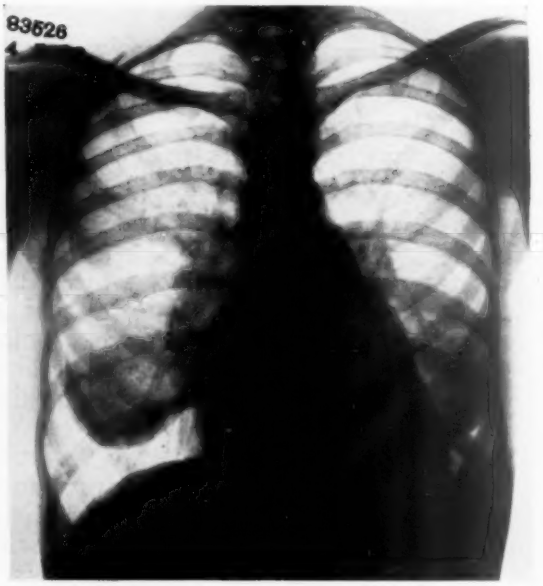



FIG. 2. Mitral stenosis and insufficiency in female aged twenty-six. The cardiac shadow shows the typical mitral deformity. There is a marked increase in the root shadows on both sides, with definite increased density along the linear markings extending well out from the periphery of the lung. This represents a case in which the changes in the lungs are not marked.

### THE PATHOLOGICAL AND RADIOGRAPHIC FINDINGS

Before discussing the radiographic findings in cardiac pneumofibrosis, it is desirable to review its pathology.

According to MacCallum,[6] macroscopically the lungs are rather pale, sometimes dry, sometimes edematous, dense, elastic and of a distinct rusty brown color.

Microscopically the capillaries in the alveolar walls become greatly dilated and tortuous, so that they project in loops into the alveolar cavities. The alveolar epithelium and other cells are desquamated into the air cells; fluid exudes from the tense capillaries, often with red blood-corpuscles. The smooth muscle in the septa that forms the vestibules in each lobule is greatly increased in bulk, and the alveolar walls in extreme cases become thickened and indurated by the appearance of new connective tissue (brown induration of the lungs).

The extent of change from the normal as seen with the *x*-rays varies widely, depending upon the severity and duration of the disease. Early changes are seen at the hilus, and as the process progresses they extend outward along the bronchial tree into the lung fields. In advanced cases the entire lung area may show these changes. The characteristic finding is an increase in size and density of the hilus shadow. The borders of this shadow are hazy and indefinite. Radiating outward from it, the lung markings are increased in width and density, but without beading. The surrounding lung tissue is less radiant than normal so that the markings appear hazy and indefinitely defined. These changes may be accompanied by irregular areas of even density of rather large size, and indefinite borders which are most frequently seen in the lower part of the chest around the descending bronchi. There is also occasional coarse mottling between the shadows of the bronchial tree. The outline of the diaphragm is usually well defined but its respiratory movements are frequently limited. The size and shape of the heart shadow are of considerable importance in arriving at a correct diagnosis, as the condition in the lungs is due to failure of the pulmonary circulation. The lung findings are invariably accompanied by an abnormal appearance of the cardiac shadow, the mitral configuration being the most common.

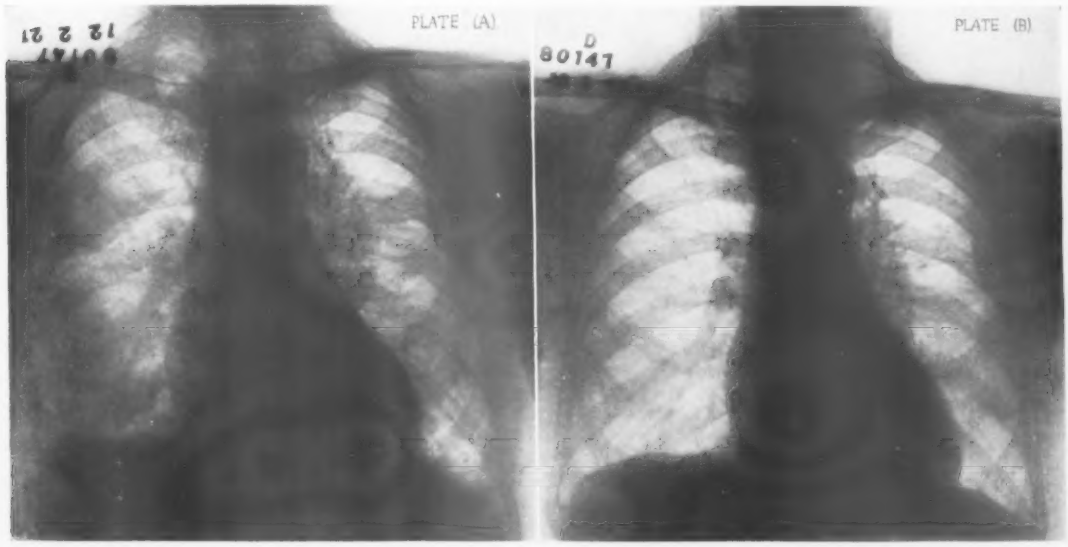



FIG. 3*a*. Cardiosclerosis, auricular fibrillation, myocardial insufficiency, with extensive changes in the lungs. Physical examination showed coarse moist rales at both bases, and dullness and diminished breath sounds at the right base. The plate has the general hazy appearance characteristic of cardiac pneumofibrosis, with the marked increase in the hilus shadows and general thickening along the course of the bronchi. The unusual appearance in this case is the areas of sharply defined increased density in the right chest and the high flat diaphragm on the right side. This could be interpreted as being due to changes in the pleura.

FIG. 3*b*. Cardiosclerosis, auricular fibrillation, myocardial insufficiency, with extensive changes in the lungs. This plate, taken of the patient six days later, shows complete disappearance of the areas of density in the right lung. At this time, the patient's cardiac condition had returned to normal.

DIFFERENTIAL DIAGNOSIS

The lung picture of cardiac pneumofibrosis is likely to be confused with dust pneumofibrosis, metastatic carcinoma and tuberculosis.

After reaching the axillary nodes and the muscular aponeurosis, mammary cancer usually becomes generalized and nearly any tissue in the body may become involved.

The lungs are reached superficially from the pleural lymphatics, while from the bronchial nodes extensions pass along the lymphatics of the blood-vessels. Extension to lungs through the blood-vessels is rare.

In the radiograph, the lungs in malignancy present several distinct types which have been described by Carman, Pfahler, Crane and others.

The type with which we are interested is that in which the deep mediastinal glands are involved, and the process spreads outward along the bronchial tree.

In the enlargement of the hilus shadow due to malignancy, the shadows are likely to be more irregular and more sharply defined. The distribution along the bronchial tree is more irregular and the process is not likely to be more marked in the lower than in the upper lobes. The heart is usually of normal size and contour.

In dust pneumofibrosis, the macroscopic picture varies according to the character and quantity of the dust to which the lungs are exposed; and the amount of fibrous tissue apparent on the cut surface of a lung depends on the length of time the process has been going on. In the early stages, practically none can be seen by the naked eye; in the advanced stages, the smaller vessels and bronchioles are distended and stand out, surrounded by dense fibrosis, in which are embedded the dust particles. In this stage, the lungs may show emphysematous blebs, bronchiectatic dilations, and collapsed areas. One of the most striking features of the gross anatomy is the black glands at the hilus and the formation of nodules in the lymph channels.

The essential differences as seen on the radiograph are the more even distribution

FIG. 4*a*. Mitral stenosis and aortic regurgitation with pulmonary tuberculosis. In the clinical picture, the cardiac condition predominated and the patient apparently died a cardiac death. In this plate there is marked increase in the hilus areas and a general haziness and thickening of the lung markings characteristic of pneumofibrosis. The process extends into the apex on the right side, and there is an area of markedly increased density near the right root.

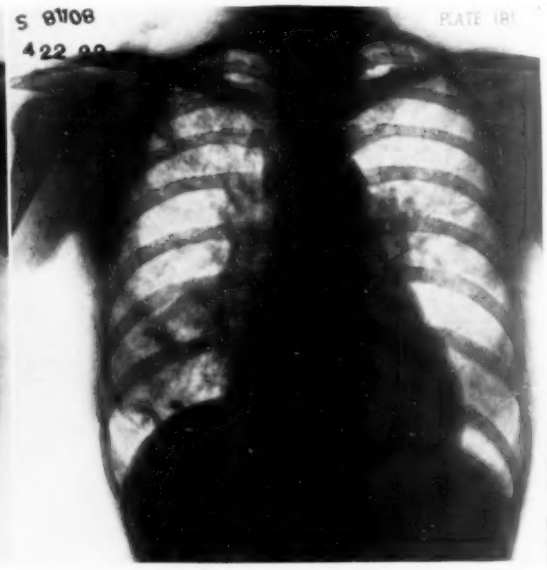



FIG. 4*b*. Mitral stenosis and aortic regurgitation with pulmonary tuberculosis. This plate, made of the patient three months later, shows a distinct increase of the process at the apex and a suggestion of cavity formation in the dull area at the base. At this time tubercle bacilli were found in the sputum.

of the densities throughout the lung fields, the absence of marked enlargement of the hilus shadow and the normal cardiac shadow.

Aside from the pneumonic patches following bronchial transportation, there is a great variety of anatomical lesions in the lung which is due to the passage of tubercle bacilli along the lymphatics or blood-vessels.

When the infection is through the lymphatic channels the bacilli produce tubercles in strings or clusters along the interlobar septum in the bronchial walls or in the walls of the blood-vessels. It is this type which is most likely to be confused with cardiac pneumofibrosis. In tuberculosis there is more definite beading and the markings are more sharply outlined. The general haziness of the lung field is absent. The cardiac shadow may or may not be normal. Ring-like shadows or areas of diminished density due to cavity formation and retraction of the mediastinal contents may be present. These latter findings are never seen in the fibrosis of cardiac lesions.

In general, the differentiation of cardiac pneumofibrosis from other forms of pneumofibrosis will depend upon the study of the cardiac shadow, the general hazy appearance of the lung field and the absence of evidence of destruction and retraction in the lungs. In this disease, as in most lung conditions, a correct interpretation of the radiographic findings will depend usually upon a knowledge of the clinical history and physical examination.

The following histories are illustrative of the cases studied:

Case I. (No. 85,097.) Female, aged fourteen.

*Clinical Diagnosis.* Rheumatic heart disease with mitral stenosis? Consolidation at right base.

*Present Illness.* May 11, 1922. One year ago, patient was sick for one month with pneumonia. Ever since she has had marked dyspnea on exertion. Has had no cough, pain or edema. No history of tonsillitis, rheumatic fever or chorea. Has tachycardia on exercise. She has been able to continue with her school work.

*Personal History.* Tonsillectomy and adenoidectomy five years ago.

*Physical Examination.* Thin, pale and only fairly well developed. Throat and teeth are negative. Pulse is 100 and is regular in rate and rhythm. *Heart.* Left border dulness is 10.5 cm. in the fifth space. $P_2$ is accentuated. No murmurs or thrills in aortic area. In the mitral region, there is a systolic and presystolic murmur and a sharp first sound; a thrill could be felt. *Lungs.* There is slight dulness and

Fig. 5. A case of metastatic malignant disease of the lung from a carcinoma of the bladder. The plate shows the irregular increase in the hilus shadow and larger markings occasionally seen in this disease. Note that the shadows are more irregular and more sharply defined than in cardiac pneumofibrosis.

squeaky rales at the right back from the base to just below the angle of the scapula. *Abdomen.* No fluid; liver dulness from fifth space to costal margin. *Extremities.* No edema.

*Note from Cardiac Clinic.* May 18, 1922. A case of rheumatic heart disease with mitral stenosis. Respiratory infections, chronic, following pneumonia (?) of tuberculosis.

Appears to be more than a chronic passive congestion. ℞ To have repeated sputum examinations and temperature taken morning and evening. Referred to pulmonary clinic.

May 26, 1922. No tubercle bacilli found in the sputum at three different examinations. Patient has had no fever. The case appears to be one of cardiopulmonary fibrosis.

*X-Ray Findings.* May 13, 1922. Thickening at the lung roots without evidence of calcification, and increase in density of the larger bronchial markings. Apices and periphery are relatively clear. Appearance may be due to lymphatic engorgement and pulmonary congestion secondary to the heart condition. Prominence of right auricular arc particularly marked in oblique view. Left ventricular arc flattened, apex pointed and displaced downward. Enlargement of heart particularly in region of auricles suggesting predominant mitral lesion.

Case II. (No. 83,526.) Female, aged twenty-six.

*Clinical Diagnosis.* Mitral stenosis and insufficiency—aortic insufficiency—secondary anemia.

*Consultation Clinic.* Apr. 6, 1922. Dyspnea and precordial pain.

*Present Illness.* In 1918, four years ago, patient was sick for five weeks with double pneumonia. After this illness, she tired easily, had a persistent unproductive cough, and dyspnea on even the slightest exerotin. Has had no edema or palpitation.

*Family History.* No history of exposure to tuberculosis.

*Personal History.* Always well until her attack of pneumonia five years ago.

*Physical Examination. Chest.* Lungs clear. *Heart.* Left border dulness, 9 cm.; right border, 4 cm. Sounds regular, slow and forceful. $P_2$ and $A_2$ accentuated. Short presystolic murmur at left apex and a diastolic at left sternal border. *Abdomen.* Negative.

*Summary.* A fairly well compensated cardiac entering hospital because of dyspnea and precordial pain. The signs are those of mitral stenosis with moderate degree of aortic regurgitation.

*X-Ray Findings.* Apr. 7, 1922. The outline of the diaphragm is well defined on the right; a little hazy on the left. Its movements are distinctly limited on both sides. The heart shadow is increased in all diameters, the greatest increase being across the base. There is marked prominence of the shadow of the auricles, particularly the left, giving the heart the typical mitral shape. The hilus shadows are very much increased both in size and density on both sides. All of the larger lung markings are also increased in size and density. Both lung fields have a general hazy appearance. The apices are clear.

Case III. (No. 80,147.) Male, aged sixty.

*Clinical Diagnosis.* Cardiosclerosis—auricular fibrillation—myocardial insufficiency.

*Consultation Clinic.* Nov. 30, 1921. Dyspnea and palpitation.

*Present Illness.* Four months ago, after an operation for hemorrhoids, had sudden shortness of breath. Because of the increasing dyspnea, he was forced to give up his work after one week. Has had no edema. Has had a sense of constriction with dull pain in the left chest, shoulder and arm.

*Present History.* "Always well and strong." Syphilitic rash in 1887. No history of tuberculosis in family.

*Physical Examination. Chest.* There are coarse moist rales at both bases, and dulness, diminished breath and voice sounds at the right base. (?) of slight dulness at the left apex posteriorly. *Heart.* Left border dulness, 10 cm. Sounds rapid and irregular. No murmurs or thrills. Marked pulse deficit. *Abdomen.* Liver felt 1 cm. below costal borders, otherwise negative.

Dec. 7, 1921. Patient suddenly returned to normal rhythm, Discharged to local medical doctor.

*X-Ray Findings.* Dec. 2, 1921. The hilus shadows and lung markings are prominent. Both lung fields are less radiable than normal. Lateral half of right chest shows homogeneous density from the second rib to base. The median border of this area has a rather well defined wavy outline. Both diaphragmatic shadows are irregular in outline and markedly restricted in their respiratory excursion. Both costophrenic angles are obliterated. There are dense white lines extending outward from the hilus toward the chest wall. No marked change at extreme bases. Appearance is not that of tuberculosis. Heart shadow is enlarged; supracardiac dulness is slightly increased.

Enlargement of heart is more to the left than right. There is definite prominence of the ascending aorta.

Dec. 8, 1921. The shadows described in the right chest have entirely disappeared. Diaphragm is still high on the right side with partial obliteration of the costophrenic angle. Markings in the lung are generally thickened.

Case IV. (No. 81,108). Female, aged thirty.

*Clinical Diagnosis.* Pulmonary tuberculosis—mitral stenosis—aortic regurgitation.

*Present History.* Apr. 20, 1922. Patient felt perfectly well and had no dyspnea or cough until onset of present illness—health always good.

*Present Illness.* On Jan. 2, 1922, while stepping off a street car, she suddenly coughed and spit up about a tablespoonful of bright red blood. There was no pain. She continued work, and one week later she coughed up blood again (ounces) five successive times in one hour. Two weeks ago she caught cold and had hacking cough with thick clear sputum since. Not breathless, but she states she climbs not more than one flight of steps without resting; no night sweats, chills or fever, no gastrointestinal or genitourinary symptoms.

*Family History.* She has had no exposure to tuberculosis.

*Physical Examination.* Extremities and lips are cold and blue. In no apparent respiratory distress. *Glands.* Moderate sized mass of freely movable non-tender glands in left neck; also large axillary glands. *Lungs.* The apices are retracted. Over the left apex, there is diminished tactile fremitus and the breath sounds are bronchovesicular to bronchial. Over the right apex, there is bronchial breathing and increased vocal fremitus. There are sticky rales over both backs. *Heart.* $P_2$ and $A_2$ accentuated; diastolic murmur at apex with harsh systolic. In the aortic area, there is a systolic ending in a loud $A_2$, with a high-pitched diastolic-presystolic thrill at apex. Blood pressure is 90/100. *Abdomen.* The liver edge could not be felt. Spleen felt 1 cm. below costal border. There are no masses or fluid.

*Summary.* Hemoptysis four months ago the initial symptom, but had no cardiac symptoms, though she was found to be a well-marked mitral stenosis. For past two weeks, she had cough and large amount of sputum. The physical examition shows loud diastolic and presystolic murmur at apex and a harsh systolic murmur at the base with a diastolic murmur along the left sternal border. This case appears to be mitral stenosis with aortic stenosis and regurgitation. The lungs are filled with sticky rales posteriorly, which did not appear to be due to pulmonary edema, for she was able to lie down with complete comfort. It is an infectious process which is very suggestive of tuberculosis.

May 5, 1922. The social service reports that the patient took a long walk on April 29th and became very weak and dyspneic. Two days later, she was found unconscious in the morning and died fifteen minutes later.

*X-Ray Findings.* Jan. 7, 1922. The diaphragmatic shadows are regular in outline. The diaphragm moves equally but poorly. The chest is of the ptotic type. There is coarse mottled dulness along the bronchi on both sides, but it is most marked in the right lower chest. In the right upper lobe, the changes extend to the apex. The process seems to be bronchial and peribronchial in type. In some plates a suggestion of dilated bronchi can be seen. There is no definite evidence of cavity formation. The heart shadow is distinctly enlarged, the increase being almost entirely downward and to the left in the region of the ventricle. There is an extensive pathological process in both lungs.

Jan. 13, 1922. The findings are practically the same as previously recorded. There is a definite process in the right lung which is more evident in the lower than in the upper lobe.

Apr. 22, 1922. There is a definite increase in the hilum shadow on both sides. The apices do not light up. Both upper lobes appear mottled, the right more than the left. At the right base there is a ring-like shadow with dense periphery and light central area and irregular increased density in the surrounding lung. There is a sharp upper border in the region of the

interlobar septum. The right diaphragm is fixed; the left, free. The process has extended rapidly since the previous observation. It now shows evidence suggesting cavity formation in the lower right side.

BIBLIOGRAPHY

1. Barjon, F. Radio-Diagnosis of Pleuro-Pulmonary Affections. English translation by James A. Honeij. New Haven, 1918.
2. Overend, Walker. The Radiography of the Chest. Vol. I., Pulmonary Tuberculosis. St. Louis, 1920.
3. von Assmann, Herbert. Die röntgen-diagnostik der inneren erkrankungen. Leipzig, 1921.
4. Stillman, Ralph G. Passive Congestions of the Lungs. Nelson Loose-leaf Med., New York, p. 468.
5. Robinson. Nelson Loose-leaf Med., New York, p. 468.
6. MacCallum. Mechanical effects of experimental mitral stenosis and insufficiency. *Johns Hopkins Hosp. Bull.*, 1906, pp. 17, 260.

# DISCUSSION ON PAPERS OF DRS. HALL, CHILDS, HOLMES AND DANN*

Dr. Moore. These two papers interest me very much. I was particularly interested in the papers of Dr. Hall and Dr. Childs about malignant growths within the chest. In 1915 I reported a series of 70 cases of secondary malignant growths within the chest. At that time we considered practically only the nodular type of metastasis. We were impressed at that time with the relative haziness of the clinical picture of secondary malignancy within the lungs. My series differs from Dr. Hall's in that we had only one case in which there was no history of blood-tinged sputum. Later we began to realize that there was a different type of secondary malignancy—the metastatic type in which we got the shower of malignant cells in the lungs. Our attention has been called to the fibrous type of metastasis. I have not been able, so far, to differentiate that from fibrosis produced by numerous other conditions.

In regard to the miliary type, I must say I find it very confusing at times to differentiate from tuberculosis.

Regarding mediastinal shadows, of course, one must rely largely on the fluoroscope to exclude aneurism. We must remember, however, that if the aneurism sack is filled with clotted blood, we will not get pulsation. We should look upon tumor masses extending from the mediastinum as probably malignant. Lymphosarcoma is the most common condition found. Primary carcinoma is the most confusing we have to deal with. When it occurs as a discrete area of increased density radiating from the hila, we can advance the opinion that it is primary malignancy. We used to think it more common in the lower lobes, but we now feel it can be found anywhere.

We have recently been confronted by a condition which is unique to me, i.e., yeast infection in the lung which simulates almost exactly our conception of what primary carcinoma should be.

Dr. Holmes' paper is a distinct contribution to literature. I have felt, personally, that there are cardiac conditions in which we meet with congestive conditions in the lung, either diffuse or localized, which are difficult to classify or diagnose.

Personally, in speaking of peribronchial tuberculosis, I think we do not make a diagnosis of peribronchial tuberculosis without consolidation in the periphery of the lung.

I was so glad to hear Dr. Holmes say that the pathologist at the Massachusetts General Hospital has put the brakes down on syphilis of the lung. I have been looking for one for twelve years.

I would ask Dr. Holmes if he found any cases of pneumofibrosis in exophthalmic goiter. We see shadows in the goiter which are difficult to explain. I would like to ask how many cases of tuberculosis he found in exophthalmic goiter.

Dr. Pirie. I would like to ask Dr. Holmes if he has really seen good clear lungs in chronic cases of heart disease.

Dr. Hickey. In cases where you have massive effusion on one side and where you suspect new growth of lung underneath effusion, if you draw off the fluid and pump the chest full of air, you will get a plate that will help you with diagnosis.

Dr. Holmes (closing discussion). In regard to the changes at the hila, I have noticed such conditions.

In treating a considerable number of goiter cases, we took plates of the chest as a routine procedure. At that time we were having considerable difficulty in differentiating the appearance from hilus tuberculosis. Since having the metabolism test, that thing has not come up.

In regard to Dr. Pirie's question, I find it rather difficult to answer. I have not gone through and studied carefully all the cardiac

* Papers read at the Twenty-third Annual Meeting of The American Roentgen Ray Society, Los Angeles, Calif., Sept. 12–16, 1922. Those of Drs. Hall and Childs appeared in March, and the paper by Drs. Holmes and Dann precedes this discussion.

cases, but I can see where that would happen. A large percentage of cardiac cases are decompensated, and it is difficult to get a good plate of the cardiac region.

The next to the last case I presented shows, I think, that it clears up when the heart is doing the work it should. A man may have a definite cardiac lesion, not of long standing, and if not decompensated, there is no change in the lung.

# A STUDY OF LOBAR PNEUMONIA AND ITS PULMONARY COMPLICATIONS BY SERIAL ROENTGENOGRAPHIC EXAMINATION*

BY L. R. SANTE, M.D.

Assistant Professor of Roentgenology, St. Louis University Medical School; Roentgenologist to St. Louis City Hospital; Consulting Roentgenologist Koch Hospital for Tuberculosis. From the Department of Roentgenology, City Hospital

ST. LOUIS, MISSOURI

Among 272 cases of frank lobar pneumonia treated at the St. Louis City Hospital during the past two years, 152 were subjected to roentgenographic examination. Roentgenograms were made at from one- to three-day intervals during the course of the disease, and the findings were carefully correlated with clinical history and physical signs in many of the cases. This study of lobar pneumonia was undertaken with a view of determining the natural course of the disease, its complications and its sequelae.

From the pathological viewpoint, four different stages of the pulmonary lesion are recognized in pneumonia: (1) The stage of congestion and engorgement; (2) the stage of red hepatization; (3) the stage of grey hepatization, and (4) the stage of resolution. The stage of congestion is characterized by the engorgement of the blood-vessels and lymphatics and the production of an active edema in the air spaces, and, to some extent, in the interstitial tissue. At autopsy, particles of such material, although considerably denser than normal, will still float on water, showing that they are still air-containing to some extent. The increase in density of this material would hardly be sufficient to cast as dense a shadow in the roentgenogram as that produced by the stages of red and grey hepatization. This stage is so transitory, however, often lasting but a few hours, and the difference in density of the shadow in the roentgenogram so slight, that it is quite obvious that any differentiation of the active stage of consolidation would usually be impossible from the roentgenogram. After the crisis has occurred, however, in the stage of resolution, the irregularly resolving and absorbing exudate produces an uneven mottled appearance in the roentgenogram, which is quite distinctive of this stage of the disease. Since, from a practical standpoint, it is, therefore, impossible to determine definitely the active stage of the disease from the character of the shadow, let us consider the distribution of the consolidation and its bearing on the course, diagnosis and prognosis of the disease.

*Roentgenographic Characteristics.* The pathologist tells us that the process of consolidation is closely confined to one or more lobes, so that a definite knowledge is necessary of the position which these lobes occupy and the location of their interlobar septa. To illustrate with more accuracy the location of the interlobar septa, this case has been introduced (Fig. 1). The inflammatory reaction incident to a pulmonary abscess in the hilus region has resulted in the pronounced thickening of all of the interlobar septa. This case represents, then, a much more practical demonstration than any which could be obtained in the autopsy room or by inspection of dissecting-room material, and is excellently adapted for a study of the interlobar septa and the position of the various lobes of the lung. From this roentgenogram, taken in the lateral position, it will be seen that the upper lobe makes up a much greater part

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922.

of the lung tissue than is generally supposed. When the tube is centered at the 5th dorsal vertebra at 28 in. distance, it will be noted that the central ray falls very closely along the interlobar fissure of the upper and middle lobes (Fig. 2A), so that the resulting shadow of upper lobe consolidation should be clear-cut and sharply outlined at its lower border, and the entire shadow should be very dense. Any slight variation in centering or tube distance

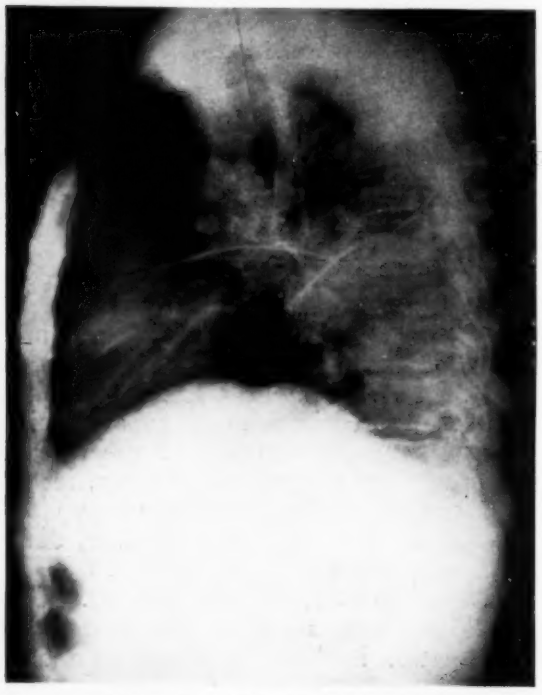


FIG. 1. Thickening of the interlobar pleura incident to a lung abscess in the hilus region. The marked thickening of the interlobar pleura serves to show the relationship of the various lobes. Note that the middle lobe does not touch the posterior chest wall at any point, but presents entirely at anterior wall.

should make little difference in the resulting shadow. Whereas middle lobe involvement (Fig. 2B) should present a similar sharpness of outline at its upper boundary and a hazy outline for its lower border, the entire shadow should not be as dense as the shadow of upper-lobe consolidation. Where the lower lobe is involved (Fig. 2C), the extreme lower portion of the lung will also be involved, and the upper border of the shadow should be more or less hazy in the ordinary position of the *x*-ray tube. When these essential points are borne in mind, a lobar consolidation involving one or more lobes can be readily differentiated and the roentgenographic characteristics easily explained.

*Course of the Disease.* Having analyzed the probable result of roentgenographic examination of consolidation of the various lobes, let us examine roentgenograms of various lobe consolidations, and pass to a

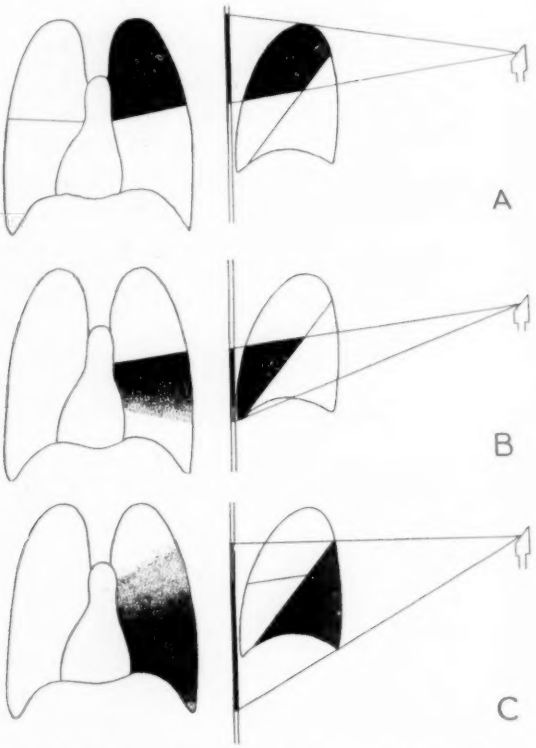



FIG. 2. Diagrams showing shadows produced by consolidation of various lobes, with the tube at 28 in. distance and centered at about the 5th dorsal vertebra. (A) Upper-lobe consolidation. The shadow is dense and the lower border is sharply defined. (B) Middle-lobe consolidation. In involvement of the middle lobe, the upper border of the consolidation is sharply defined, the lower portion is hazy. The extent of the shadow produced is relatively small compared to consolidation of other lobes. (C) Lower-lobe consolidation. The upper border of the shadow is hazy and the extent of the shadow produced is greater than any other lobe. The costophrenic angle often remains aerated during the acute stage of the disease.

consideration of the course of the disease. It will be seen that in practice the roentgenographic findings conform closely to those anticipated; atypical borders and anomalous lobes may occasionally be encountered (Fig. 3, A, B, C).

In a great many of our cases there was complete consolidation of an entire lobe

at the first examination, even when the first roentgenogram was made within twenty-four to forty-eight hours after the onset. It is quite evident, therefore, that in many cases, probably the greater majority, complete consolidation occurs within a very short time. In a certain number of instances, however, the pathological process does not progress so rapidly. In some of these patients, on first examination, a definite consolidation was seen at the hilus region, which, on successive examinations, progressed rapidly outward, rarely requiring more than twenty-four to forty-eight hours to reach the periphery (Fig. 4). Whether of the slower, progres-

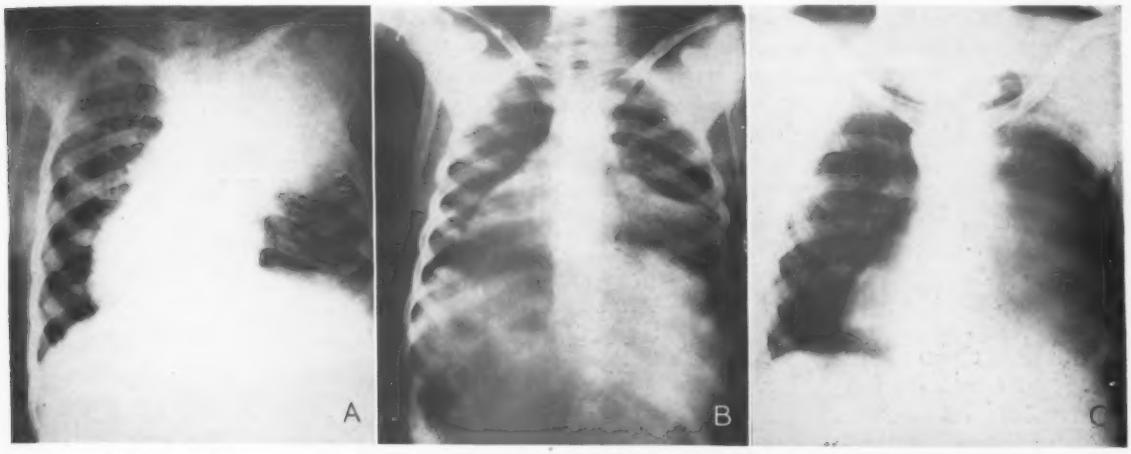



FIG. 3. (A) Upper-lobe consolidation. Note sharply defined lower border. (B) Middle-lobe consolidation. Note sharply defined upper border, hazy lower portion and small extent of involvement. (C) Lower-lobe consoldiation. Note hazy upper border, and large amount of involvement. Lower-lobe consolidation extends higher than middle-lobe consolidation. The costophrenic angle is aerated.

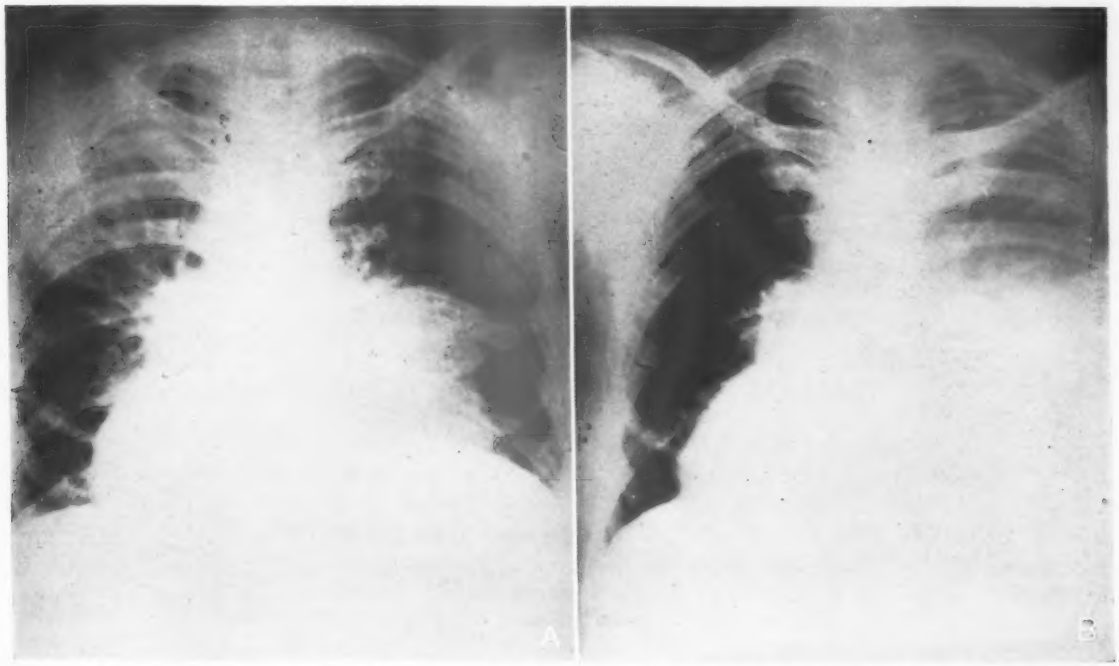


FIG. 4. Lobar pneumonia. (A) Consolidation commencing in the hilus region. (B) Plate made twenty-four hours later, showing extension to the periphery and involvement of the entire lower lobe.

sive type, or the rapidly consolidating form, the shadow produced is of a homogeneous character, presenting an even density throughout, showing only dense peribronchial markings, which, at the onset of the process, are usually seen through the consolidation shadow. The zone of advance of the process is feathery and uneven, and shades off into the normal tissue. The edge of the advancing shadow is composed of the radiating extension of accentuated peribronchial markings. This is probably due to the fact that the process, commencing in the hilus area, has progressed more fully to dense consolidation than that in the periphery, which is involved in the later stage, and also because the amount of consolidated tissue obstructing the ray is greater in the inner than in the outer zone of the lung. As the disease progresses, and consolidation becomes more dense, the peribronchial markings become obliterated, and the entire shadow may be of homogeneous density. In the natural course of the disease, little change is seen in the shadow from the time complete consolidation is reached until the time of the crisis. Shortly after the crisis, resolution begins, and the shadow changes rapidly from the homogeneous type to that of an uneven mottled appearance. Very soon the accentuated peribronchial markings again become apparent, and the uneven mottled area gives place to groups of soft isolated infiltrations. The entire area may be overshadowed by a pleural haze if the pleura has become thickened during the course of the disease. Resolution is very rapid, however, and all evidence of infiltration and consolidation may have completely disappeared within three days after the crisis (Fig. 5). Ordinarily, however, complete resolution requires a somewhat longer time—from seven to ten days; persistence of consolidation for fourteen days after the crisis should be viewed as distinctly pathological. It is, therefore, quite evident that the consolidation in lobar pneumonia, at least in many instances, starts at the hilus and extends peripherally.

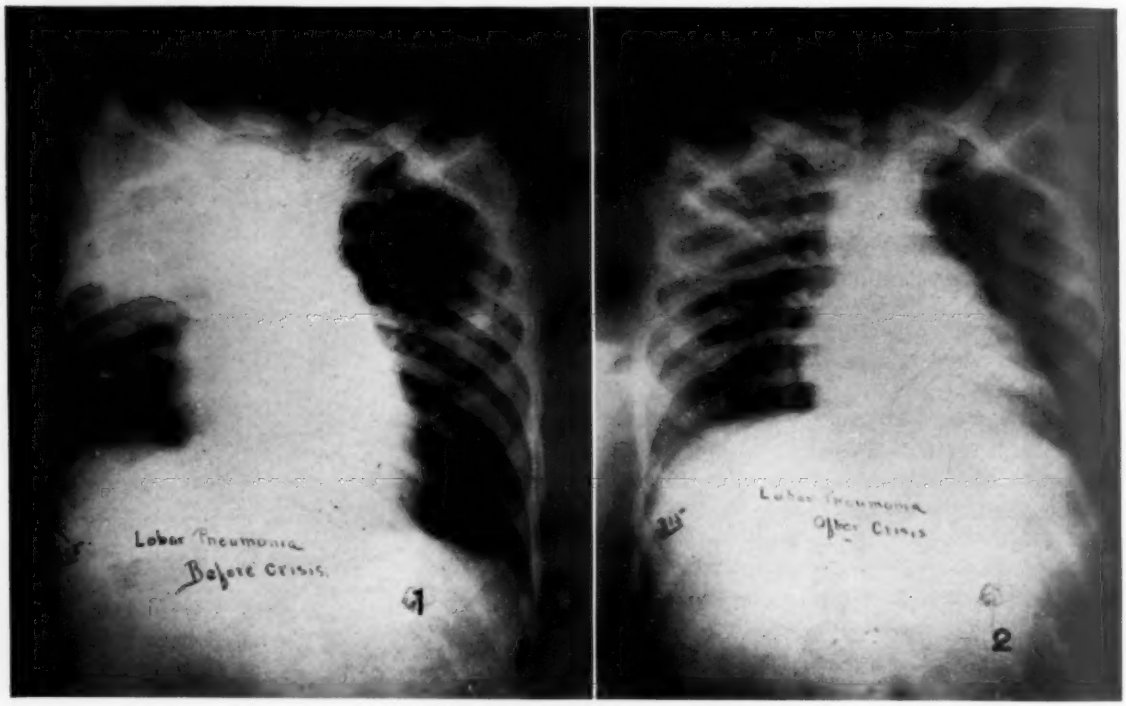



FIG. 5. (1) Upper right lobar pneumonia showing characteristic shadow. Plate made just before crisis. (2) Showing almost complete resolution in three days after crisis.

*Review of Literature and Discussion.* A review of literature, however, seems to show this at variance with the observa-

tions of some men. Mason[1] described the roentgenographic appearance of pneumonia in children as a triangular shadow with the base at the periphery and the apex at the hilus of the lung. He further stated that the consolidation commences in the periphery and gradually progresses to involve the hilus. He quotes the conclusion of Weill and Mouriquand[2] from the consideration of 350 pneumonia cases studied by *x*-rays, that the early shadow is usually triangular in shape, with its base usually conical and generally axillary. They have never seen a shadow which could be considered entirely central. Mason himself describes this consolidation and illustrates two cases, both of right upper-lobe involvement, which, in the roentgenograms, suggest a more intense consolidation at the periphery. He emphasizes the observation that the process of consolidation always begins in the part nearest the pleura and thus, during the beginning of the process, at any rate, is separated from the root of the lung by normal aerated lung tissue, and that central pneumonia, therefore, does not exist. On the basis of roentgenograms taken in cases in which physical signs of consolidation did not exist, where there was apparently an aerated zone of normal lung tissue beneath the surface consolidation, he concludes that in these instances the lack of physical signs is due to the failure of transmission of the breath sounds from the hilus through the aerated lung tissue to the surface. In December of the same year, Stewart,[3] in a publication on the differential diagnosis of lobar pneumonia and empyema in children, comments on Mason's theory as follows: "While I am not prepared at the present time to accept in its entirety the explanation given by Dr. Howard Mason of New York for the frequent lack of positive physical signs in some cases of pneumonia, I do believe that his theory holds good in many cases showing late physical signs. As many cases of pneumonia are believed to start at the root and spread toward the cortex, this explanation would seem to apply only to those commencing at the pleural surface." In the same publication he describes a root or hilus pneumonia in children, and shows illustrations of this type of consolidation beginning in the hilus region of the lung, spreading fanshaped into all the lobes and usually remaining confined to this region. In spite of this demonstration of root pneumonia, Barjon,[4] in his book on Pleuro-Pulmonary Affections, says: "These proofs do away with the idea of central pneumonia which would explain the late appearance of physical signs. Indeed, the shadow of the pneumonic triangle begins at this base, and this base is always cortical, since it develops in the axilla. Finally, the radioscope never shows in any case a primary central focus without some relation to the cortical portion of the lung. In short, central pneumonia does not exist, but everything progresses as if it did." In a recent publication the writer[5] reported 12 cases of hilus pneumonia occurring among 276 cases of pneumonia examined. Eleven of these were of the distinctly inflammatory type, 1 occurring in a child, and 10 in adults. In a previous publication,[6] the existence of hilus pneumonia in the adult in connection with the influenza epidemic was pointed out.

It is quite probable that many cases of pneumonia in children do follow the course indicated by Mason in his essay on pneumonia: beginning at the periphery and progressing inward to the hilus. Among the cases included in this series several instances are present where the consolidations occurring in children followed this course (Fig. 6). To say that this is the only course followed in children is manifestly wrong, in view of the many existing cases of hilus pneumonia previously cited in the literature. It is also unmistakably true that many cases do occur in adults in which the consolidation starts in the hilus region and spreads toward the periphery. While it is possible that in adults, certain cases may exist in which consolidation starts in the periphery, we have never encountered one in which this relationship could be established. Mason's explanation of the failure to elicit certain physical signs is probably quite correct in certain instances. It is also true that a similar obscurity in physical signs can occur when the consolidation is confined to the hilus region, in so-called hilus or root pneumonia, and it is

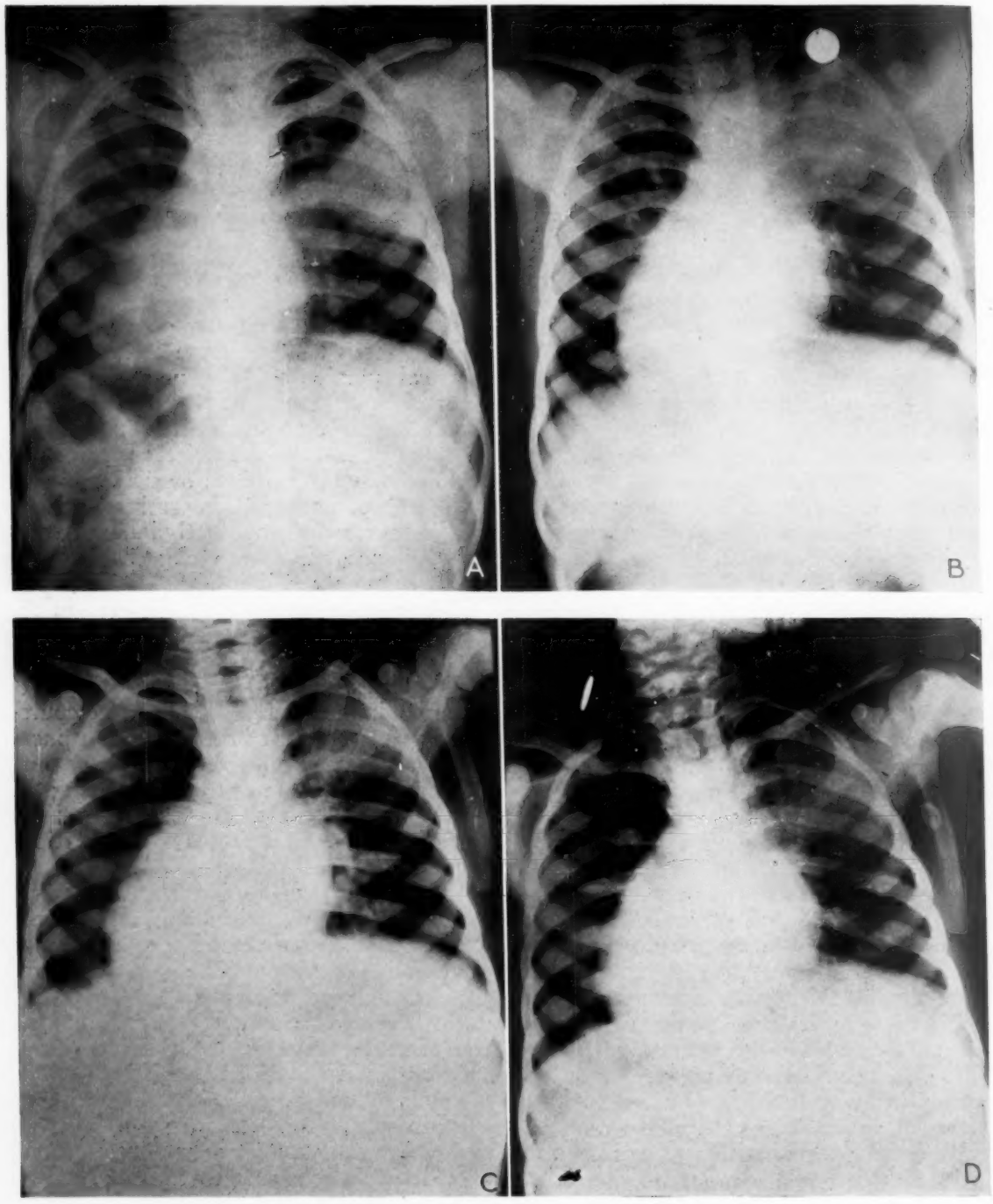



FIG. 6. Lobar pneumonia right upper lobe. Type referred to by Mason as starting in the periphery and advancing to hilus. (A) Note the consolidation of lower and outer portion of the right upper lobe. The sharply-defined lower border establishes the identity of the lesion as upper rather than middle lobe consolidation. (B) Within forty-eight hours there was advancement of the process to involve the entire upper lobe. (C) Shortly after the crisis resolution begins. (D) Continued favorable resolution of the process.

probable that the same explanation holds in these cases. Furthermore, from the plates which have been shown of lobar pneumonia in adults, it is evident that, in the greater number of instances, lobar pneumonia starts as a central consolidation in the hilus region and spreads peripherally. It would seem, therefore, that, at least in the majority of instances, our old conception of lobar pneumonia was the correct one; the obscurity in physical signs in certain instances being due to a centrally located consolidation which had not yet reached the surface.

*Prognosis.* Pneumonia, uncomplicated by other pulmonary lesions, may itself prove fatal. Neither the location of the consolidation, its extent nor the density of the consolidation is a determining sign in the prognosis. The most extensive involvement of lung tissue may end in recovery: the smallest areas of involvement may result fatally. The process in one lobe may resolve only to be followed by consolidation in another, possibly adjacent to the primary involved lobe, possibly remote. All the lung tissue on one side may be involved in a massive consolidation. As a general rule, however, involvement of a single lobe gives much the best prognosis, especially where the shadow shows the characteristic changes described as natural for the course of the disease. Usually, lobar pneumonia resolves very rapidly: three days after the crises may be sufficient for complete resolution; seven to ten days, however, is the average time. If fourteen days elapse after the crisis without signs of progressive favorable resolution, or if no crisis occurs within this period, the condition is abnormal, and it can be depended upon that some complication of pneumonia is present. Persistence of the shadow over this period did not occur in a single instance, unless there was some complicating pathology.

*Pulmonary Complications.* The most frequent pulmonary complications of lobar pneumonia are:

1. Dry pleurisy with thickening of pleura.
2. Pleural effusions, serous or purulent; either general or localized.
3. Plastic serofibrinous pleurisy.
4. Chronic interstitial pneumonia or fibrosis.
5. Lung abscess.

Dry pleurisy, resulting in a thickening of the pleura, both at the periphery and in the interlobe, is so frequent an attendant with pneumonia that it is probably best considered as a part of the pathological process. The presence of a pronounced pleural reaction is, in itself, almost pathognomonic of a recent inflammatory lesion. Roentgenographically, the linear shadow

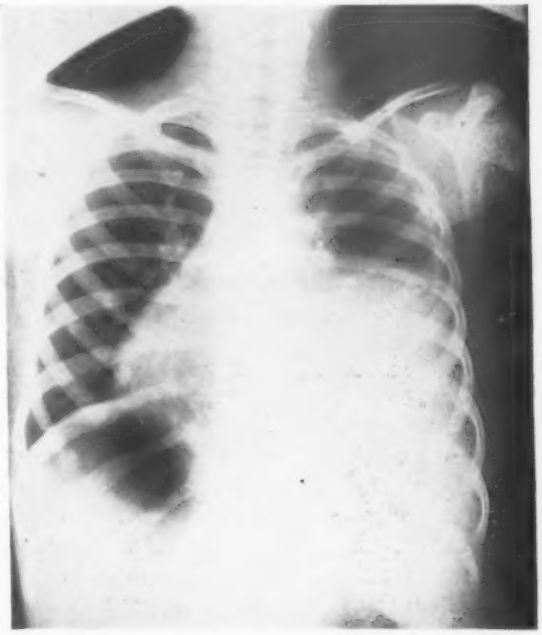


FIG. 7. Very frequently a small amount of fluid forms in the pleural cavity during the course of the disease. Note the ribbon-like shadow running along the parietal wall. The rounded appearance of the upper portion of the consolidated area is suggestive of a small collection of interlobar effusion.

running upward along the parietal wall of the chest and the dense linear shadow extending across the chest from the hilus to the periphery when the interlobar pleura is involved, are familiar to all roentgenologists. After the disease has progressed to involve the pleura, a small amount of serous fluid may collect (Fig. 7). This obscures the costophrenic angle and produces a ribbon-like shadow along the parietal chest wall, not unlike pleural thickening, and at times almost indistinguishable from it. This occurs so frequently during the course of the disease that it

seems quite probable that it is a provision of nature to keep the inflamed pleural surfaces apart, and prevent adhesions. Its presence is of little significance, merely adding to the difficulty of differential diagnosis between lower-lobe pneumonia and pleural effusion. Larger collections of fluid may occur in the free pleural cavity during the course of a pneumonia, and these have the same characteristics of fluid encountered under ordinary conditions in the chest. Where the pneumonic consolidation is in the upper portion of the chest, any additional shadow forming in the lower chest from the accumulation of fluid can be readily detected. The costophrenic angle, being the most dependent portion of the chest, is first to be obscured, and the shadow extends across the lower portion of the chest running up along the axillary border. The upper border of the effusion is hazy and concave, extending higher up toward the axillary side. The heart and mediastinal structures are usually displaced somewhat to the opposite side, due to the weight of the fluid. This is not an infallible sign, however, and has not been observed on certain occasions with moderately large effusions. Where a primary consolidation in the lower lobe is complicated by a pleural effusion, the diagnosis becomes more difficult, and it is often very hard to decide just how much of the shadow is due to consolidation and how much to fluid. Pleural effusions encountered as a complication of pneumonia may be either serous, serofibrinous or purulent. Since it is impossible to tell from the density of the shadow the character of the fluid, it is impossible to differentiate between serous effusion and empyema. The clinical picture may give some indication, but aspiration is the only reliable test. Pleural effusion may not only be general, involving the entire chest cavity as previously described, but may also be localized by adhesions between the two pleural layers at any point at which the pleural layers come in contact with each other (Fig. 8). This may be between lobes of the lung, when an interlobar effusion results. Such effusions usually present a clear-cut shadow extending across the entire half of the chest from the hilus to the periphery.

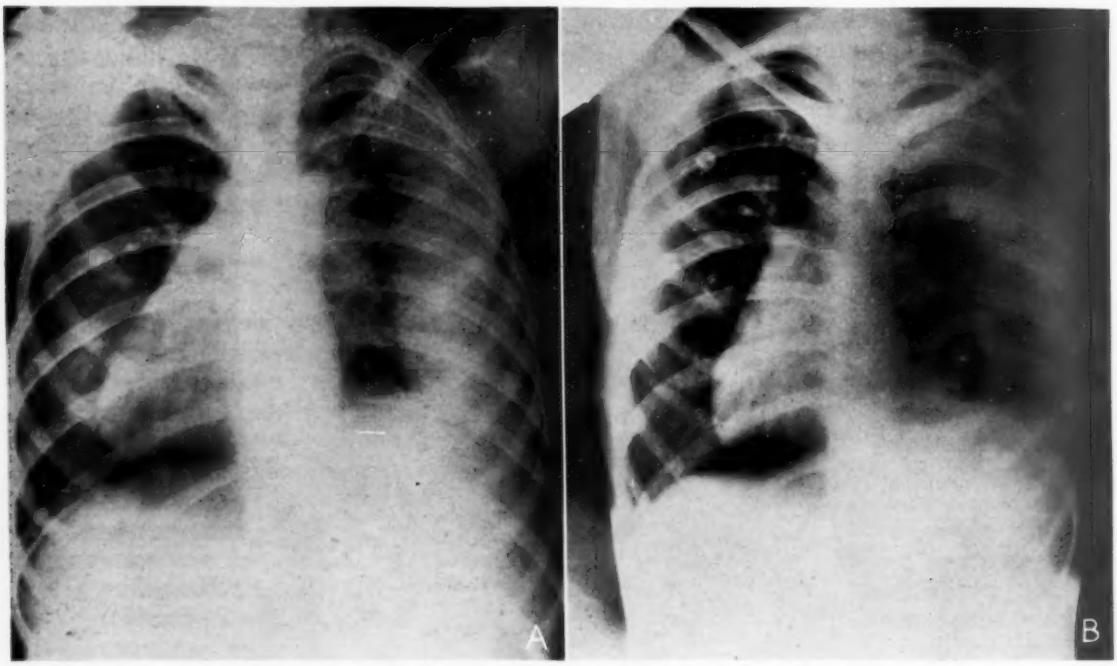



FIG. 8. Localized fluid entrapped between the two pleural layers. Localized collections of fluid, unassociated with plastic serofibrinous pleurisy, are sharply outlined. Being between the layers of pleura, the shadow is peripheral in location. (A) Localized fluid without pocketing. (B) Localized fluid showing a tendency to pocket formation.

The outer margin is often rounded and the lower border sharply defined, in these respects differing from consolidation of the middle lobe (Fig. 9). Collections of fluid, localized by adhesions of the parietal and visceral pleura, may occur in any portion of the pleural cavity. They are most frequent in the lower portion of the chest, in the axillary border and in the posterior cul-de-sac. Occasionally they are observed in the upper portion. Regardless of position, localized pleural effusions are nearly always sharply outlined unless they occur in connection with plastic serofibrinous pleurisy. In determining the location of such localized effusions, the lateral view is most helpful. Such effusions may be either serous or purulent in character. That small collections of pus, either in the lung or entrapped between the pleural layers, frequently escape detection, is evidenced by this case (Fig. 10, A, B, C, D). In this instance two drams of thick pus were aspirated from the lower chest during the height of the consolidation. Resolution progressed favorably and complete restoration to normal resulted. Pneumothorax is a very rare complication of pneumonia, and usually occurs in connection with post-pneumonic empyema.

Another condition which sometimes complicates pneumonia is plastic serofibrinous pleurisy. This complication, although not so frequently met with, is apt to be more serious than the other complications previously mentioned. Presenting no features akin to an effusion, it must necessarily be considered as a separate entity from the roentgenologic standpoint. The pneumonic process may run a natural course ending by crisis, the temperature may fall to normal and remain so for a short time, only to be followed by a post-critical rise and a persistent septic temperature, or a definite crisis may never occur, the temperature gradually assuming this septic character. Roentgenographically, the consolidated area does not resolve in the customary manner (Fig. 11). Large blotchy areas of increased density remain usually most pronounced at the periphery, the central portion of the shadow showing indication of normal resolution. The pleura is extremely thick and presents a stringy appearance, due to the fibrinous exudate. At autopsy the pleural surface is shaggy, with heavy strands of fibrinous exudate enmeshing small abscesses. These abscesses may attain considerable size, and may even represent localized areas of effusion. The pus content is usually very thick and creamy, due to the high fibrin content. The process may invade the lung, resulting in multiple small abscesses and interstitial fibrosis. Such involvement may be small in extent and may completely resolve, even

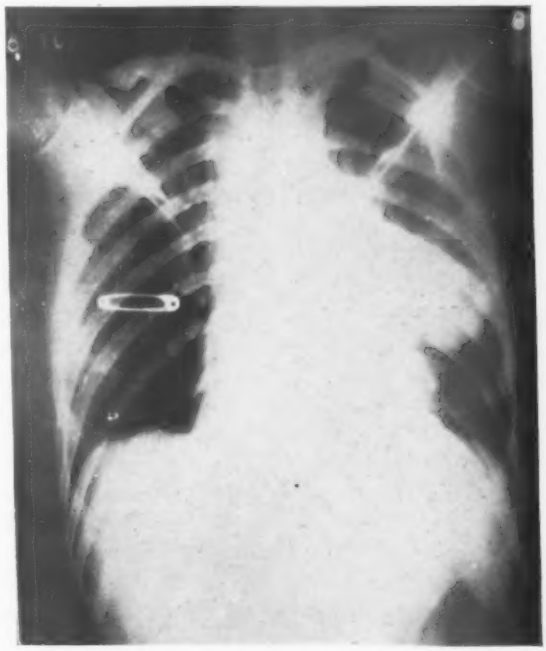


FIG. 9. Interlobar collection of fluid. Note the dense homogeneous shadow extending from the hilus to the periphery; note also the rounded appearance of the outer margin.

where pus is present, leaving only a small pleural adhesion. Resolution of such a process usually requires six to eight weeks, however. Repeated roentgenographic examinations will indicate the progress of resolution, and will aid in determining the prognosis. Where the condition is very extensive, however, the outlook for ultimate complete restoration to normal is not so good. Where any considerable amount of pus is present, or where it is evident from repeated roentgenographic examinations that the condition is not progressing favorably, surgical intervention and evacuation of the pus may aid materially in the

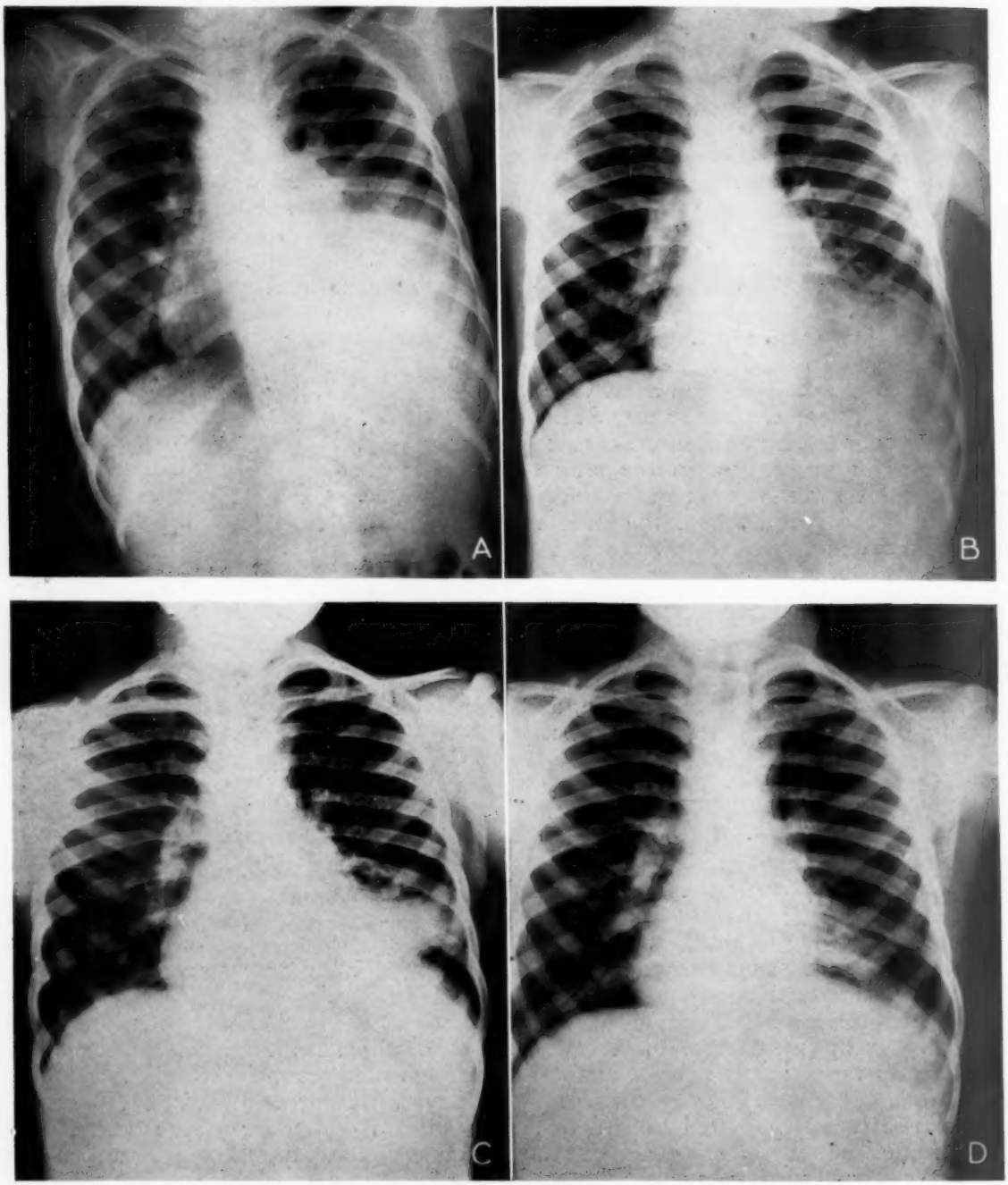



FIG. 10. That small collections of pus, either in the lung or entrapped between the layers of pleura, often escape detection, is evidenced by this case. (A) Lower right-lobe pneumonia just after crisis. At this time a needle was inserted and two drams of pus aspirated from somewhere within the involved area. (B) Plate made three days after aspiration of pus. Note the network appearance of upper border and marked recession of the process. (C) Plate made three weeks later, showing continued favorable but very slow resolution. (D) Plate two weeks later showing continued favorable resolution. Almost complete restoration to normal. Clinically the child had fully recovered.

ultimate recovery. By reason of the enmeshed character of the pus and its very viscid consistency, it often happens that surgical intervention or any other method of treatment is of little avail, and the process goes on to ultimate fibrosis (Fig. 12). The pleura, both parietal and visceral, becomes enormously thickened, obliterating the pleural cavity. The small abscesses entrapped between the layers of pleura throughout the fibrinous exudate become absorbed and are replaced by fibrous tissue.

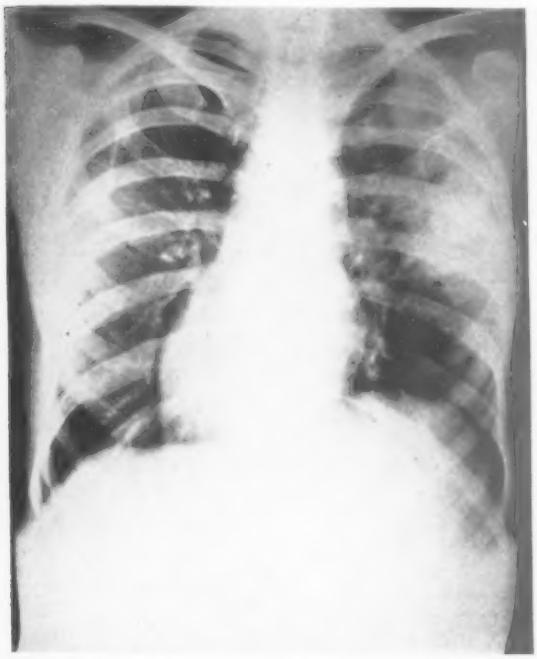


FIG. 11. Plastic serofibrinous pleurisy with localized collection of fluid. Note the peripheral location of the shadow, and that when associated with plastic pleurisy, localized collections of fluid are not sharply outlined.

FIG. 12. Chronic interstitial pneumonia following organization of a plastic serofibrinous pleurisy. Note the narrowing of the interspaces, the elevation of the diaphragm and the displacement of the heart and mediastinal structures to the right.

Organization takes place, and finally scar tissue is formed. The lung tissue may become involved and an interstitial fibrosis, similar in character, may result, either from an extension of the process, or as a result of functionless condition produced in the lung. The ultimate stage of the process is a contraction of the scar tissue. The heart and mediastinal structures are drawn over toward the affected side. The diaphragm is elevated and the intercostal spaces are narrowed. The entire side of the chest becomes more shallow than the normal side, a condition which is even more apparent since compensatory emphysema of the normal lung usually results. The entire lung is practically replaced by fibrous tissue showing a dense irregular shadow. The condition produced is known pathologically as chronic interstitial pneumonia. Some modification of this process is probably what has been referred to as "unresolved pneumonia."

The last complication of pneumonia to which I desire to call attention is lung abscess. Persistence of the consolidated area beyond the time normal for a lobar pneumonia, without favorable signs of resolution, is almost pathognomonic of abscess formation. Often, the diagnosis of a complicating abscess can be made by the roentgenographic appearance, fully five to seven days before the indications are present clinically.

*Differential Diagnosis.* During the early stage of lobar pneumonia, the consolidation may be confined to the hilus region and resemble, in all respects, hilus pneumonia. Within twenty-four hours, however, the hilus consolidation should spread toward

the periphery, confining itself to a single lobe, if the condition is due to lobar pneumonia. If the condition is due to hilus pneumonia, the shadow will remain constant and will not spread peripherally. As a general rule, when the consolidation is fully developed, there is little difficulty in the diagnosis of lobar pneumonia. Occasionally, however, even the acute shadow of lobar consolidation may be confused with other conditions. Caseous tuberculous pneumonia (Fig. 14) may cause a mas-

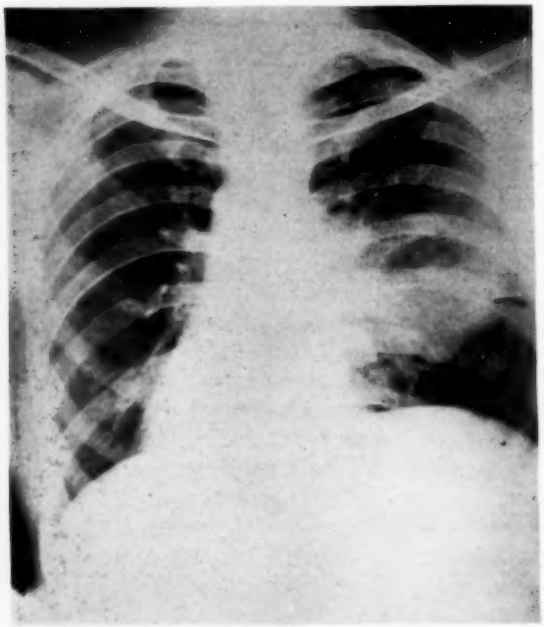


FIG. 13. Lung abscess following lobar pneumonia of the lower lobe.

sive homogeneous consolidation confined closely to one or more lobes, which may resemble in every detail the consolidation from lobar pneumonia. The symptoms and course of the disease readily differentiate the conditions. Caseous tuberculous pneumonia requires from three to four months for resolution, whereas lobar pneumonia may completely resolve in as many days. Caseous tuberculous pneumonia usually leaves behind definite cavity formation; lobar pneumonia resolves completely and leaves a perfectly normal appearance. It is undoubtedly true that caseous tuberculous pneumonia may occur in any of the lobes of the lung, but the upper lobes are undoubtedly the most frequent site; the right side being most frequently involved. Where the roentgenographic evidence alone is analyzed, the differential diagnosis may depend entirely upon the course of the disease. The clinical history, however, may serve at once as a determining factor. In the tuberculous type of pneumonic consolidation, the temperature is never so high and there are morning remissions even to normal, a condition not obtained in lobar pneumonia. There is not the pronounced leucocytosis obtained in lobar pneumonia. The patient does not feel exceptionally bad, and may even be up and about; in pneumonia, on the other hand, the patients are quite ill and confined to bed. In this connection may I say that in the opinion of the writer, positive diagnoses as to chest conditions without a previous consideration of the clinical history are very hazardous.

Only under the rarest circumstances would the lobar distribution of a bronchopneumonia be confused with lobar pneumonia. The comparative rarity of the former condition, its occurrence in connection with septicemia or as a complication of influenza, its invasion by peribronchial infiltration and the blotchy, uneven appearance of the shadow will aid in the differential diagnosis of the two conditions.

Syphilis of the lung produces, at times, massive homogeneous consolidation, which may, at a single examination, resemble lobar pneumonia. While the consolidation from syphilis is usually more massive, a similar picture may be presented by lobar pneumonia, and again the determining factor must be sought in the clinical history. Whereas one would expect a patient with lobar pneumonia of this extent to be desperately ill, syphilis of this extent may have remarkably few symptoms. The Wassermann reaction and resolution of the consolidation after salvarsan are the deciding factors.

Tumors of the lung rarely produce any confusion. The only type of new growth which, in the writer's experience, has ever in any way simulated pneumonia, is the metastasis from a hypernephroma. Hypernephroma (Fig. 15) may be confined to the lower lobe of the lung produc-

ing a dense homogeneous consolidation which at times may simulate lobar pneumonia or fluid. Three such cases have come under the writer's observation, which were either confirmed by autopsy or microscopic sections from material obtained by lung puncture.

One of the most difficult differential diagnoses may be in the differentiation between lower-lobe pneumonia and fluid, or the simultaneous existence of both. The upper border of a pleural effusion extends from the hilus region upward and outward to the axilla, and may conform very closely to the upper border of a consolidation in the lower lobe. It is true that the costophrenic angle is obliterated in fluid and is usually well aerated during the acute stage of lower-lobe consolidation, but very often a somewhat later stage of the disease finds it obliterated, whether by extension of the consolidation to this remote portion of the lung, or by obliteration from a small collection of protective fluid, makes little difference in the difficulty of diagnosis. The heart and mediastinal structures are usually displaced in fluid, but, as has been said, that is not an infallible sign, for either no displacement at all may be present, or it may be so slight as to be indeterminate. Under certain conditions, therefore, the differential diagnosis between lower-lobe pneumonia and fluid becomes very difficult. A method which we have used occasionally, and have found most useful in the differentiation of the two conditions will be briefly outlined (Fig. 16). Since the upper bounding plane of the lower lobe is on a relatively straight line running from behind forward and downward, it follows that the image cast on a plate of consolidations of the lower lobe, if the ray is projected along the upper surface, will be a definite straight line, clear-cut at its margin and very dense. When made in the ordinary position, the beam of *x*-rays traverses the wedge-shaped upper portion of the lower lobe, causing a gradual shading off into the normal lung, and resulting in a hazy upper border which is not sharply outlined and not dense. Fluid, on the other hand,

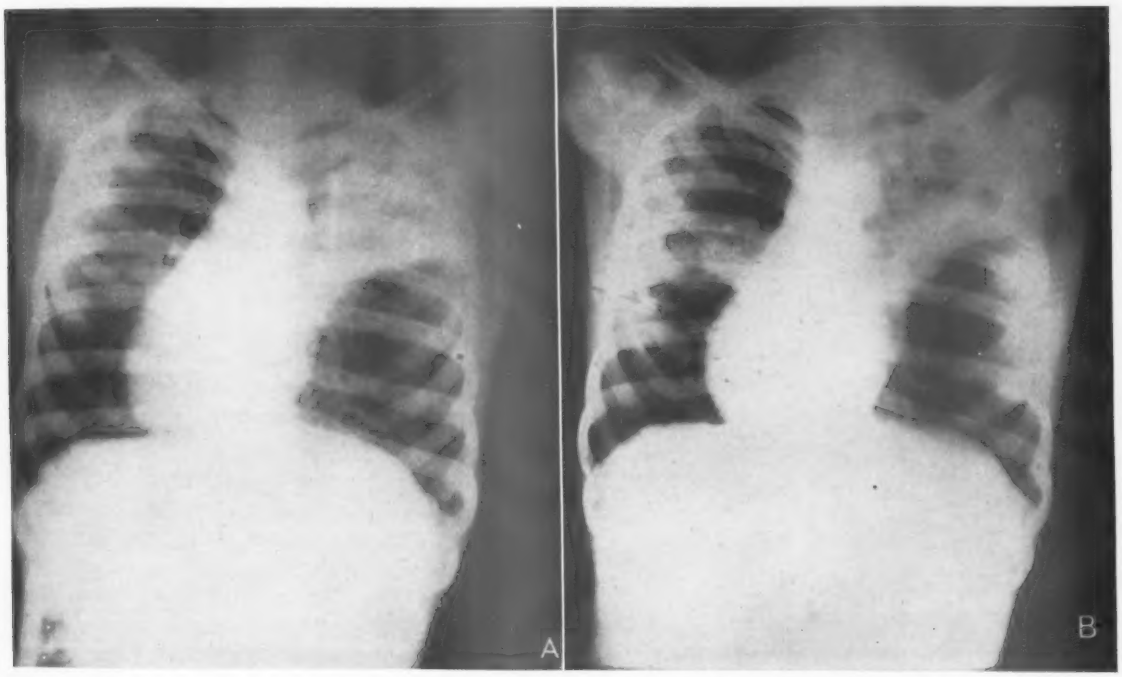



FIG. 14. (A) Acute caseous tuberculous pneumonia of the lobar type. This condition simulates very closely the consolidation from lobar pneumonia, and can only be differentiated at the onset by the lack of serious clinical symptoms, and later on by the protracted course of the disease. (B) Three months later. Same resolution has occurred with cavity formation.]

when it has reached a stage confusing it with pneumonia, owing to the curved line which it produces, both from before backward and upward, and from within outward and upward, never casts a clear-cut upper border, no matter what the position of the tube. While roentgenograms made in the ordinary position may show a hazy, indefinite upper border, either in lower-lobe consolidation or in fluid, roentgenograms made with the tube centered high up will show a clear-cut upper border if the shadow is due to consolidation, but a persistently hazy outline if it be due to fluid.

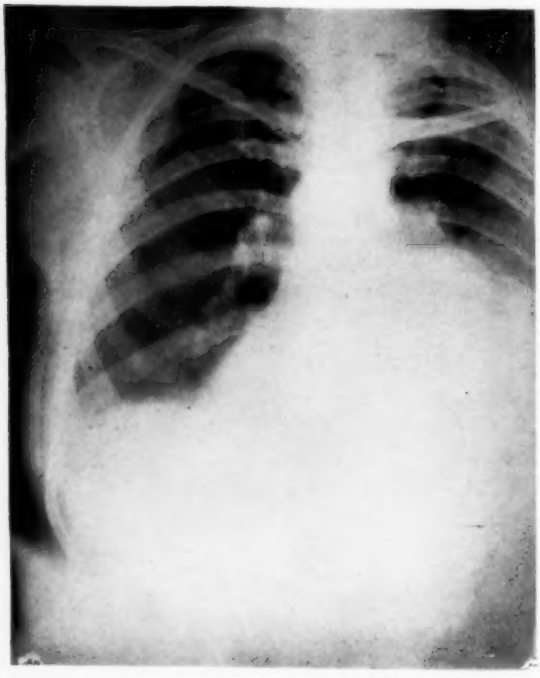


FIG. 15. Hypernephroma, metastatic in the chest, causing consolidation of the right lower portion of the chest simulating lobar pneumonia or fluid, and may be indistinguishable from these conditions without clinical history.

In the resolving stage, the most easily confused lesion is that of the infiltrative type of tuberculosis. This is especially the case where the pneumonia involves the upper lobe. Here, again, the clinical history may be the determining factor: a short duration and profound illness suggest an inflammatory process; a longer duration without serious illness labels the condition as more probably of tuberculous origin.

Localized pleural effusion will rarely be confused with tumors of the pleura, as the history and previous plates made during the course of the disease will differentiate. Localized pleural effusions are sharply outlined, and, being between the layers of the pleura, are seen at the periphery rather than at the midportion of the lung. In these respects they differ from lung abscess, which is not clearly outlined, being surrounded by an irregular zone of inflammatory infiltration; being in the lung substance, its shadow appears as a consolidation and usually does not extend to the periphery.

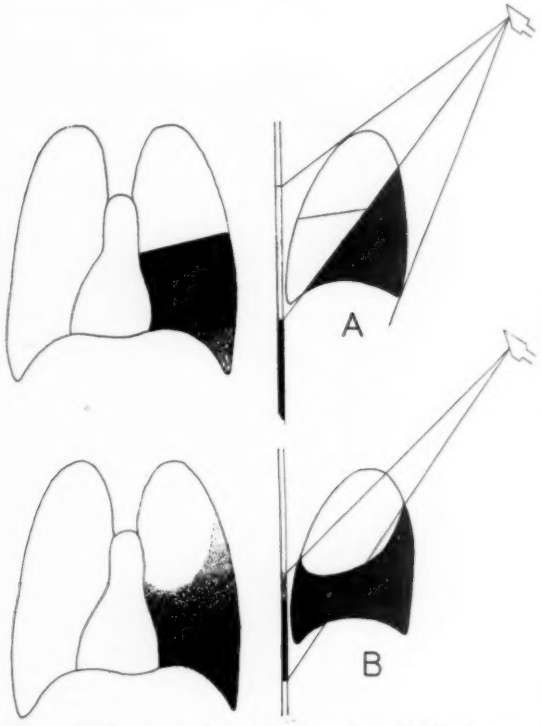



FIG. 16. Diagram showing character of shadows cast by lower-lobe consolidation and fluid with high position of tube. In this position the upper border of lower-lobe consolidation becomes sharply defined and the upper border of fluid remains hazy, no matter what the position of the tube.

Plastic pleurisy with localized collections of pus has the same peripheral location, differing thus from abscess of the lung, but is not sharply outlined and differs in this respect from an ordinary pleural collection.

The differentiation of organized pleurisy and interstitial pneumonia from large pleural effusions or massive pneumonic

consolidations lies in the manifestations of scar-tissue contraction—the pulling over of the mediastinal structures, the pulling up of the diaphragm, the narrowing of the intercostal spaces and decrease in size of the pleural cavity on that side.

## SUMMARY

1. Owing to the similarity in appearance, differentiation between the stages of active consolidation in lobar pneumonia is impossible from the roentgenogram.

2. In the majority of cases, lobar pneumonia starts as a consolidation in the hilus region, rapidly spreading peripherally, and involving an entire lobe. In a few cases in children, the onset of consolidation is cortical and progresses toward the hilus.

3. The shadow produced is homogeneous and is usually confined to one or more lobes. The shadow produced by involvement of the various lobes is indicated by diagrams.

4. During the stage of resolution the shadow becomes mottled and irregular, complete resolution being effected often in a very short time—three days.

5. The average time for resolution is seven to ten days after the crisis. Persistence of shadow or failure of resolution after fourteen days is distinctly pathological, and suggests some complicating lesion.

6. The pulmonary complications most frequently encountered following pneumonia are:

(*a*) Dry pleurisy with thickening of the pleura.

(*b*) Pleural effusion, either serous or purulent, and either general or local.

(*c*) Plastic serofibrinous pleurisy.

(*d*) Chronic interstitial pneumonia or fibrosis.

(*e*) Lung abscess.

7. Their roentgenographic differentiation is indicated.

## BIBLIOGRAPHY

1. Mason, H. H. Lobar pneumonia in childhood. *Am. J. Dis. Child.*, March, 1916.

2. Weill and Mouriquand. *Am. de méd. et chir. inf.*, xvii, 275.

3. Stewart, Wm. H. Am. J. Roentgenol., December, 1916, p. 557.

4. Barjon, F. Radio-Diagnosis of Pleuro-Pulmonary Affections. New Haven, 1918, p. 103.

5. Sante, L. R. Study of hilus pneumonia by serial radiographic examination. *J. Rad.*, 1922, iii, 6.

6. Sante, L. R. Study of influenza pneumonia by serial radiographic examination. *J. Missouri M. Assn.*, February, 1921, xviii, 43.

## DISCUSSION

Dr. Ullmann. Dr. Sante has covered the subject so thoroughly that I am rather at a loss what to say. At Santa Barbara we do not have very much pneumonia, although we have plenty of other things.

I would like to ask Dr. Sante if he has seen at any time what Dr. Billings spoke of in 1916 as pseudo-lobar pneumonia. I do not know whether there were any roentgen examinations made of these cases or not.

Dr. Sante did not speak of purulent bronchitis as a complication. I saw one child, twelve years old, who, after a pneumonia had cleared up, continued to run a temperature. The question of any empyema was seriously considered, although the physical signs were pretty well gone. Stereoscopic plates showed a cast of the entire bronchial tree which appeared as though it had been injected with an opaque medium. This condition entirely cleared up three or four days later.

I was glad to hear the statement made that any shadow remaining over two weeks was abnormal.

There was a condition seen during the war following gassing, especially after mustard gas, which I have never seen in civil life. I would like to ask if anyone else has seen it. During my service with the British Army I was fortunate enough to make stereoscopic plates of men dying with acute pneumonia following inhalation of mustard gas. These plates were made immediately after death and an autopsy held within a half-hour. I can best describe the appearance as a honeycomb. Bleeding into the pleural cavity was found at autopsy and the lungs were filled with patchy emphysematous areas in an edematous lung. These areas of emphysema and edema were apparently responsible for the honeycomb appearance in the roentgenograms. The hemorrhage and emphysema were probably due to the violent coughing which was such a marked feature of these cases.

I would like to ask Dr. Sante if there was any routine position for taking pneumonia cases. I take it that a portable outfit was used. I would like to know if he found it difficult to have the patients sit up for the examination.

DR. LEWALD. I was present at the time the paper referred to by Dr. Sante was read, and at that time made the statement that I had seen 2 cases of pneumonia starting at the root and extending to the periphery. This bears out what Dr. Sante has also observed, and I am sure that his observation is quite correct; that we do have pneumonia starting at the hilum and later extending out to the surface, although that may not be as common as pneumonia starting at the surface and extending to the root.

DR. SANTE (closing discussion). Referring to Dr. Ullman's statement as to psuedo-lobar pneumonia; one of the cases which I presented was such a case—so-called lobar distribution of brochopneumonia. Autopsy proved it to be bronchopneumonia following an influenza infection.

In the army service we were afforded opportunity to examine a great many influenza pneumonia cases at daily intervals, and were able to follow their development from clusters of peribronchial infiltration to the complete lobar lesion. Without serial radiographs the differentiation from lobar pneumonia would be very difficult.

I have had no personal experience with membranous bronchitis or recently gassed cases.

I was very glad indeed to hear of Dr. LeWald's observations and am quite sure that many here have had a similar experience. I feel, however, that the vast majority of lobar pneumonia cases start at the hilus and progress to the periphery. In a great number of instances this progress is very rapid, but many times a case will be encountered while the consolidation is still in the hilus region. In these patients serial radiographic examinations will show the progress of the lesion to the periphery.

# PROGNOSIS IN TUBERCULOSIS OF THE LUNGS FROM EXAMINATION BY THE X-RAYS*

BY A. HOWARD PIRIE, M.D.

MONTREAL, CANADA

WHEN a roentgenologist makes a diagnosis of pulmonary tuberculosis, it is seldom that he sees the patient after a lapse of six months, so that he can confirm his report. If he makes a mistake and gives a positive diagnosis and the patient is well after a year, no blame is attached to the roentgenologist and the physician in charge gets the credit of a cure. If, on the other hand, he makes a mistaken negative diagnosis and the patient goes from bad to worse, the patient naturally goes elsewhere for another *x*-ray report. It thus happens that our reports on pulmonary tuberculosis are not checked up as they are on fractures. We learn it very quickly if we make a mistaken diagnosis in stomach or bone work, but not if we give a mistaken diagnosis in a case of pulmonary tuberculosis.

In order to check up my own diagnoses of pulmonary tuberculosis I have analyzed my findings during three and a half years at a soldiers' hospital. This hospital has had the opportunity of following T. B. cases for three to four years, and during that time I examined 2,321 chest cases, making 2,574 stereoscopic plates. Of these, 141 were cases of T. B. of the lungs. Some of these cases reported once or twice a year for *x*-ray examination. All are now routinely examined by stereoscopic sets.

I selected the films of patients who had pulmonary tuberculosis and who were examined by *x*-rays year by year, and put these films up side by side. By this means I have been able to see the advance in the disease from year to year. From the excellent clinical notes which have been kept of these patients, I have confirmed the clinical findings with the *x*-ray plates and made a short summary to go with the plates. It is interesting to note that in general the *x*-ray findings of pulmonary tuberculous disease have been in advance of the clinical findings. I have also been struck by the fact that patients in whom the *x*-rays have shown no tuberculosis have been sent to a sanatorium and have returned after about a year with a report

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922. Discussion of this paper and the others in the same symposium will appear in a later number of the JOURNAL.

of disease arrested. In these cases, on comparing *x*-ray plates made before and after the sanatorium treatment, no trace of T. B. was seen at either examination. In such a case I have felt inclined to congratulate the patient on not contracting T. B. from his companions at the sanatorium. I consider that as to sending a patient suspected of tuberculosis to live in a sanatorium, to mix with undoubted cases (if no T. B. has been found in the sputum and the *x*-rays do not give a positive diagnosis of it) that patient would be better off and run less risk at home than in a sanatorium.

My earliest diagnoses of T. B. are given from stereoscopic plates showing mottled areas. Advanced cases are easily recognized, and cases which are so early as not to show mottling I do not diagnose as T. B. Simple intensification of the bronchial tree is certainly not an early sign of tuberculosis, but may be a result of former healed tuberculosis. It is very constantly present running up to the apices when the note at the apices is flattened. This, to my mind, does not indicate early T. B., for in none of my series extending over three and a half years has this simple intensification of the bronchial tree been followed by active T. B.

I shall detail one case to you, typical of others showing the *x*-ray diagnosis preceding the clinical to the extent of one year.

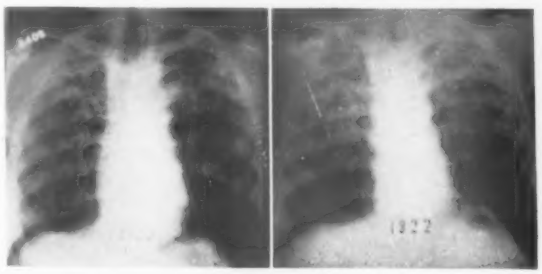
CASE I. Male, complained four years ago of varicose veins and nothing else. No *x*-ray examination made. Three years ago he complained of a cough. The clinician reported no adventitious sounds, but impaired note at both apices. *X*-rays at this time showed T. B. at left apex and a localized pneumothorax.

The next examination reported was two years ago when the clinician stated that his general condition was good: rales at right apex; otherwise breath sounds normal (1921).

The next examination was six months ago, when he had cough and expectoration, breath sounds vesicular, and a few rales in both upper lobes. A week after this examination he reported spitting of blood.

From the clinical report from 1919 to date, I would not expect that *x*-rays made in 1920, 1921 and 1922 would show that the disease had progressed. The first *x*-ray examination made in 1920 showed a distinct circular shadow like a cavity which completely disappeared in the later examinations. This circle was a localized pneumothorax.

In watching series of *x*-rays, made at intervals of a year or more, of cases of T. B., it is interesting to note that as the disease advances the heart grows smaller. This smallness of the heart has given rise to the idea that a small heart is a characteristic of a tendency to tuberculosis. I have often been struck by the absence of T. B. in cases of very small heart. I believe a small heart is the result of advanced T. B., and not a predisposing cause of T. B.




CASE II. Shows the increase of the disease and the diminution in the size of the heart over a period of two years.

CASE II illustrates this. The *x*-ray examination made in 1920 showed a fair-sized heart, while the appearance as seen in 1922, when the disease had advanced, showed the heart much smaller. In 1918 this man weighed 135 lbs., and in 1922 he weighed 105 lbs.

CASE III indicates the progress of the disease with *x*-rays made in 1919, 1920, 1921 and 1922. That in 1919 showed greater disease with small pneumothorax. The patient's weight was then 108. The pneumothorax continued for the next two years and cleared up in 1922, when his weight was 111 lbs., a gain of 3 lbs. The *x*-ray examination in 1922, however, showed the heart smaller than in 1919.

The clinical reports in 1918 state, "lungs negative," and again in 1919, "lungs negative." Not till 1921 did the

clinician note the presence of crepitations. The last clinical note in 1922 stated that the patient was generally weak, thin and pale. These films illustrate a nonprogressive type of T. B. In 1919 he was an undoubted case of T. B. from *x*-ray examination, when the clinician reported no adventitious sounds present. It illustrates the appearance and disappearance of a small localized pneumothorax.

CASE IV shows the appearance of chronic T. B., with positive sputum without active disease. There is an interval of twelve and a half months between the two *x*-ray exposures, and during that time the patient gained six pounds in weight. The later plate shows increased calcification, but the heart has grown smaller. The decrease in the size of the heart is the worst feature in this case from the *x*-ray point of view. I would call this a slowly progressive chronic T. B.

CASE V. The *x*-ray report of T. B. preceded the clinical report. The two plates were made at an interval of thirteen and a half months. In the 1921 plate no T. B. was shown. In the 1922 plate the T. B. is evident; and the decrease in the size of the heart is very marked.

CASE VI is the type in which, from the *x*-ray appearance, a bad prognosis should be given. In 1919 he showed a pneumothorax at left apex with evidence of T. B. in right and left lung. The following year the pneumothorax had increased and the heart had grown much smaller. This decrease in the size of the heart is a bad sign. The plate made in 1922 showed the heart to be a little larger than in 1920. This may be due to dilating heart from impending failure, as death occurred ten days after this last plate was made.

CASE VII indicated extensive T. B., which, after fifteen months, showed marked improvement. The 1920 plate shows T. B. in middle of right lung and at right apex. The 1922 plate shows less evidence of disease about the middle of right lung. The evidence of improvement is enhanced by the fact that the heart has not grown smaller. The patient's weight is recorded at 120 lbs. in 1918, and the same in 1922. Here, then, is a man who has lost no weight, whose heart has not grown smaller, and whose T. B. has cleared up considerably as shown by *x*-rays. This case leads me to ask if T. B. growing from the hilus (as this case appears to be) is more liable to clear up than T. B. beginning in the upper third of the lung.

CASE VIII shows that the heart of a nonprogressive T. B. does not decrease in size. He had treatment for 326 days in a sanatorium during 1917–18. His weight was 120 lbs. in 1918 and the same in 1922. The appearance of the plate of 1920 is characteristic of nonprogressive T. B. Note once more that the heart did not decrease in size in this nonprogressive case of T. B.

CASE IX shows T. B. with very little if any advance, as shown by examination over a period of twenty months, with gain in weight during that period. Both plates show a fibroid condition at the right apex, but the most important point is that the heart has not decreased in size, but is actually a little bigger in 1922 than in 1920.

CASE X was diagnosed as T. B. by an experienced roentgenologist in 1919. I consider that he made a mistaken diagnosis, as after three years there is no evidence of T. B., though the root of each lung has that appearance which makes one hesitate whether to give a positive or a negative diagnosis of T. B. He was 4 lbs. heavier in 1922 than he was in 1919. This is a case of chronic bronchitis and not T. B. The size of the heart should be noted. It is as large in 1922 as it was in 1919. Had it been progressive T. B., the heart would have grown smaller.

## RESUME

A. Favorable prognosis is based upon:
   1. Absence of abundant mottling.
   2. Presence of calcification in roots of lungs, and better still, in parenchyma of lungs.
   3. No diminution in size of heart.

B. Unfavorable prognosis is based upon:
   1. Abundant fluffy mottling.
   2. No calcification anywhere.
   3. Diminution in size of heart.

C. The diminution in size of heart with progress of the disease can be easily shown by *x*-rays. The diminution is out of proportion to the wasting of other muscles of the patient.

# ENTEROLITHS*

BY W. W. BOARDMAN, M.D.

SAN FRANCISCO, CALIFORNIA

ENTEROLITHS are rarely recognized as the cause of occasional acute and chronic gastrointestinal disturbances, until so demonstrated at operation or necropsy. Indicative of their infrequency is the fact that in many of the standard works on gastrointestinal diseases, they receive no mention, and in others, but a brief note. Fitz and Williams report but three enteroliths in 713 cases of intestinal obstruction due to extraneous bodies, and Treves but 20 enteroliths in 1,152 cases of intestinal obstruction due to various causes.

Enteroliths are of three varieties. The first and most common are the hard enteroliths of relatively high specific gravity, usually dark brown in color, spheric or oval, generally smooth, single or multiple, averaging two to three cm. in diameter, but occasionally reaching 23 cm. Section is apt to show a concentric stratification about a central organic nucleus of chalk-like or dirty white layers, frequently alternating with others of a brownish color. The composition varies somewhat, but "usually consists of carbonates and phosphates, as the phosphate of calcium, the phosphate of magnesium and ammonio-magnesium phosphate. In addition, they usually contain organic substances, including a little cholestrin and occasionally other inorganic salts, as sulphate of calcium." Second, the less frequent soft enteroliths of low specific gravity and irregular outline which are composed of thickly interlaced masses of indigestible fibers and membranes, together with a small amount of other organic and inorganic material. This variety is relatively common in Scotland as the so-called oat stone or avenolith. Third, the very rare enteroliths that follow the prolonged administration of mineral drugs—calcium carbonate, magnesia, iron and salol.

Enteroliths may originate in any part of the bowel, but are generally stated to be more frequently found in the large bowel, especially in the cecum.

The mechansim of the origin and development of the hard enteroliths is not as yet clear, for although the bowel excretes inorganic salts, and under favorable conditions these may be deposited about some foreign body nucleus, there is no satisfactory explanation for the retention of this body until it has attained such size that its spontaneous evacuation is difficult or impossible. In response to the long-continued presence and gradual enlargement of the foreign body within the lumen of the bowel, there usually follows a more or less fusiform dilatation of the involved section of bowel, together with a definite hypertrophy of the wall. The enterolith lies quite free in the center of this dilated section, but meets with obstruction at either end.

Keeping in mind the very gradual development of these foreign bodies and the equally gradual compensatory changes occurring in the bowel, the resulting functional disturbances are readily understandable. For years, no appreciable clinical manifestations may appear. In time, however, symptoms arise and, as might be expected, constipation of some degree is the commonest. Associated symptoms are loss of appetite, nausea, belching, vomiting, flatulence, abdominal distress or attacks of abdominal pain, periodic attacks of diarrhea with or without evidence of inflammatory reaction, mucus, pus and blood. These symptoms vary in severity from time to time and are the symptoms of chronic low-grade intestinal obstruction. Rarely after years of such symptoms, there suddenly develops the typical picture of an acute intestinal obstruction, demanding prompt surgical intervention. More frequently, the process continues until death occurs from some intercurrent condition or until the clinical course becomes so distressing that surgery is resorted to, usually under a mistaken diagnosis and frequently with faulty surgical procedure, such as appendectomy, cholecystectomy, gastroenterostomy, etc.

Physical examination is apt to be entirely negative, but occasionally a tumor may be felt, hard, freely movable and, at

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept 12–16 1922.

times, tender. The commonest locations are the cecum, sigmoid and rectum.

The routine laboratory investigations render little or no assistance, and, as yet, *x*-ray studies have rarely been of aid in the pre-operative diagnosis. However, if the condition be kept in mind, careful radiologic studies should more frequently be successful.

From the reported cases it is evident that some enteroliths are not of sufficient density to be recognized in the plain abdominal roentgenogram. It is also probable that enteroliths of sufficient density to be so recognized are missed by not examining an abdominal roentgenogram obtained before the administration of barium, as especially with the double-meal technique, the barium in the small bowel may readily obscure an enterolith in the sigmoid or pelvic colon.

FIG. 1. Showing the shadow cast by the masses felt in L. L. Q. and in the pelvis.

FIG. 2. Appearance after giving barium meal.

With the routine barium meal, three direct findings may appear. First, a dilated and elongated section of the bowel; second, a filling defect resulting from the varying densities of the enterolith and the barium; third, if the meal be completely followed, an intensification of the shadow cast by the enterolith resulting from barium adhering to its surface. The barium enema should demonstrate more conclusively the dilatation and elongation of the involved section if in the colon; it should also show the filling defect and the intensification of the enterolith shadow after evacuation of the enema. Pfahler recommends the inflation of the colon with gas, but in his case it was of no assistance, and it seems probable that with a properly studied barium meal and enema, such a procedure would be superfluous.

Enteroliths in the small bowel will give less definite findings, the intensification of the enterolith shadow from adhering barium being the most likely. Careful study should discover some evidence of disturbed motor functions, such as have recently been emphasized by Mills.

The enteroliths of sufficient density to be recognized on the abdominal roentgenogram can, by the above procedures, be definitely demonstrated within the intestinal lumen and thereby differentiated from calcified glands, kidney or ureteral stones, or other less dense bodies such as movable kidney, new growths, etc.

Differential diagnosis must be considered under three heads. First, in the relatively rare cases with acute intestinal obstruction, early surgical intervention will be of greater importance than the recognition of the exact etiological factor. Screen and plate studies of the abdomen before, during and after a barium enema would seem to

promise well in suitable cases. Second, in the cases with chronic abdominal symptoms presenting either a palpable mass or an abnormal opacity on the plain abdominal roentgenogram, the barium meal and enema should show their position within the lumen of the bowel. They must then be differentiated from gall-stones, fecal masses and foreign bodies, if possible. Third, in the cases presenting chronic gastrointestinal symptoms, but without palpable mass or evident abnormal opacities on abdominal plates, we must rely on a carefully executed barium study to demonstrate the enterolith and to differentiate it from the various other causes for such chronic symptoms. The difficulties here will frequently be multiplied by previous operative procedures, gastroenterostomies, etc., which tend to center the attention on one part of the gastrointestinal tract.

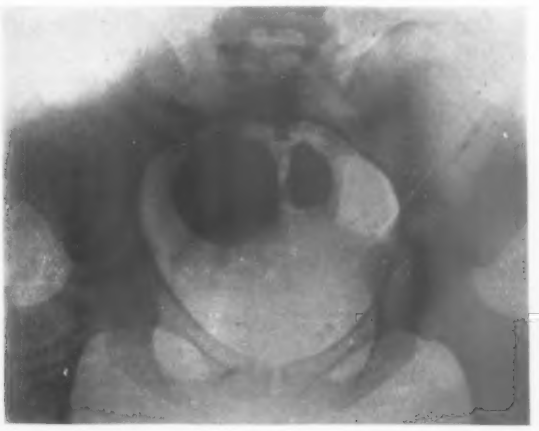


FIG. 3. Appearance after catharsis and colon irrigations.

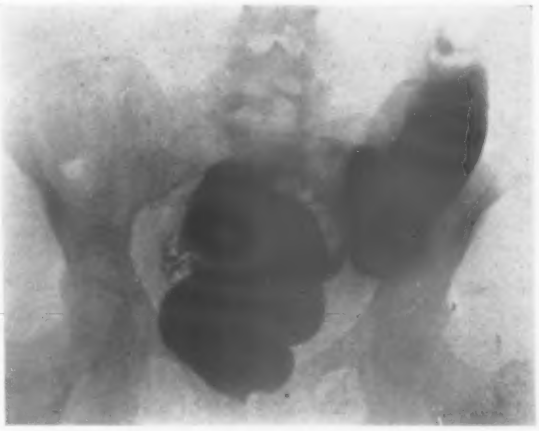


FIG. 4. After barium enema.

The following case report is typical:

Female, graduate nurse, aged twenty-seven. Came on January 14, 1921, complaining of pain in the epigastrium felt through to the back, acid regurgitation, sudden explosive and profuse vomiting without nausea, belching, loss of appetite, diffuse abdominal distention, marked borborygmus, marked flatulence, constipation alternating with diarrhea, and loss of weight.

*Family History.* Unimportant.

*Past History.* General health never good. Usual diseases of childhood. No recent acute diseases, other than a streptococcus sore throat in 1912. Respiratory, circulatory, cutaneous, nervous and urinary systems negative. Menstrual history irregular, but otherwise unimportant.

*Gastrointestinal system.* Had always had trouble, which began in infancy, with constipation, vomiting and colic, although she was breast-fed. During her early childhood days, constipation was her constant complaint, in spite of persistent treatment. At the age of seven she was having practically constant trouble with constipation, distention, distress after small amounts of food, belching, regurgitation and vomiting, marked and embarrassing borborygmus. She was underweight and undersized and lost a great deal of time from school. During her high-school period, she would regurgitate her lunch practically every day on the way back to school. She also had attacks of marked abdominal distention with crampy pains, relieved by the passage of large quantities of flatus followed by profuse and explosive diarrheal movement. Following such an attack, she would be relatively free from distention and abdominal distress for several days. Her condition continued practically unchanged until April, 1912, when an appendectomy was performed, at which time, a right-sided ovarian cyst as large as an orange was said to have been felt. Following the operation, there was some amelioration of symptoms, but no real relief. By March, 1913, all the old symptoms were back with increased severity. At this time it was customary for her to take soda six or more times at night and eventually get relief by vomiting. In

the middle of 1914, the symptoms were extremely troublesome (she lost 40 lbs. in six weeks) and finally after failing to get relief with ulcer diet and hospital attention, a posterior gastroenterostomy was done with the Murphy button. This was passed forty-nine days later.

Following the operation, she was quite comfortable for six months, when symptoms reappeared with less severity; her principal complaints at this time being constipation with flatulence, distention, regurgitation and attacks of imperative

FIG. 5. Photograph of enterolith.

diarrhea. During this period she was very careful about her diet, more especially in regard to the quantity. In 1917, she began her nurse's training and was able to complete it without loss of time, although never free from gastrointestinal distress.

In October, 1920, after six months of private night duty, all her old symptoms reappeared, epigastric sensitiveness felt through to the back, recurring attacks of colicky abdominal pain, belching, acid regurgitation, explosive vomiting, distention, increasing constipation followed by colicky attacks in which she passed large quantities of intestinal gas, followed by several imperative liquid stools. After such an attack, there was always a short period of relief.

In December, 1920, she was placed on ulcer diet with Sippy powders, after a barium meal study, which showed a functioning gastroenterostomy and an apparent cap deformity with tenderness, presumably the evidence of a duodenal ulcer. No improvement resulting, patient entered Stanford Hospital on Jan. 14, 1921, for more thorough study and treatment.

Examination at this time was negative except for diffuse abdominal tenderness, hyperchlorhydria and hypersecretion. A second *x*-ray study showed an apparently normal outline for stomach and cap, except for the gastroenterostomy. At six hours, there was a very small amount of barium remaining in the antrum and cap. Head of the meal was in the ascending colon. Ileum and cecum appeared normal. At twenty-four hours, barium was scattered through the colon. The splenic flexure was high and filled with gas.

The history and findings failing to confirm a diagnosis of ulcer, ulcer treatment was discontinued and patient was put on a bland diet—sedatives, gastric lavage, etc. A diarrhea having appeared, patient was given liberal doses of bismuth subcarbonate over several days.

All therapeutic efforts were proving of no avail, when, on Feb. 5th, the patient noticed a hard, freely movable tumor about the size of an orange in the lower left quadrant of the abdomen, very slightly tender to pressure.

Pelvic examination revealed a large hard mass lying behind the uterus and not directly connected with the mass felt in the lower left quadrant. *X*-ray examination showed two masses of slight density corresponding to the above. Barium meal markedly increased the density of these shadows and demonstrated them to be in the lower sigmoid and pelvic colon.

It was therefore, concluded that we were dealing with a fecal accumulation in the pelvic colon and sigmoid. Thorough catharsis and repeated daily colonic irrigations brought away large amounts of granular material, so that after about ten days, the lower pelvic mass had disappeared and the upper mass was considerably smaller and freely movable. Nevertheless, symptoms persisted and on discontinuing the colonic irrigations there was evidence

of re-accumulation of the material in the sigmoid and pelvic colon.

Further *x*-ray study was undertaken on Feb. 15, 1921, and demonstrated a round dense shadow about 8 cm. in diameter corresponding to the tumor mass felt. Barium enema entered readily to the middle of the sigmoid, where it surrounded the mass; this portion of the sigmoid was dilated. Descending colon apparently normal.

On Feb. 21, 1921, the abdomen was opened by Dr. F. J. Cowan through a lower mid-line incision. The pelvic organs were found normal. Within the lumen of the sigmoid there was a large hard, round, mass which could be displaced freely upward to a point about two inches from the junction of the sigmoid and descending colon and downward to within about the same distance from the rectosigmoid junction. There was a fusiform dilatation of the sigmoid, its largest central portion being about three times the normal diameter, and the walls hypertrophied. When the enterolith dropped down into the dilated curve of the sigmoid, it could be seen to exert definite traction on the rectosigmoid junction. The mechanism of the obstruction could be easily determined. The enterolith acted as a ball valve and as it was carried toward the rectosigmoid junction, it occluded the lumen of the bowel and the obstructive symptoms appeared. When it dropped back into the dilated curve of the sigmoid, relief came with the expulsion of gas and fecal matter. As the concretion could not be displaced downward into the rectum, it was removed through a transverse incision in the upper portion of the sigmoid opposite the mesosigmoid.

Postoperative course was uneventful; practically all symptoms disappeared, and at present, patient states that she has regular spontaneous bowel movements, no diarrhea, no distention, no regurgitation and no vomiting, has gained 20 lbs. in weight, and eats a regular diet, but finds that relatively small amounts satisfy her.

The enterolith was nearly spheroid in shape, of stony hardness, its surface smooth, and it measured 3 by 2¾ in. and weighed 45 gr. The sawed surface was equally dense throughout, its central portion was of a dark greenish color, while the periphery was laminated with alternate layers of dark green and dirty grey material.

*Chemical analysis.* By F. A. Cajori of the Department of Chemistry, Stanford University:

| | Per Cent |
|---|---|
| Fat and free fatty acids less than | 1.0 |
| Cholesterol | 0.8 |
| Fatty acids from soaps | 16.8 |
| Calculated as calcium stearate | 17.9 |
| Ash | 36.3 |

*Analysis of Ash:*

| | |
|---|---|
| CaO | 12.3 |
| MgO | 15.5 |
| $P_2O_5$ | 8.5 |

## CONCLUSION

1. Enteroliths, although rare, must be kept in mind in all chronic gastrointestinal cases.

2. Keeping them in mind, correct preoperative diagnosis is possible by careful physical and *x*-ray studies.

3. In all operations upon obscure gastrointestinal cases, a thorough exploration should precede operative measures.

# ROENTGEN THERAPY OF ACUTE INFECTIONS OF THE ANTRUM AND FRONTAL SINUS*

BY JOHN D. OSMOND, M.D.

CLEVELAND, OHIO

THIS paper is intended as a preliminary report of results obtained from the treatment with the *x*-ray of twelve cases of acute inflammation of the frontal sinus and antrum. Ten of the cases were in the acute purulent stage, three of which were bordering on the chronic stage. Two were in the acute catarrhal stage.

The attention of the writer was called to this subject when patients were relieved from pain in the forehead for a time after the making of radiograms for the diagnosis of a suspected frontal sinusitis.

The first treatment was given in 1916 in a case of frontal sinusitis that was steadily getting worse under the usual treatment given by the rhinologist. The anterior end of the middle turbinate had been removed and good drainage obtained. For ten days the patient complained of more pain each time she came to the office and there was no cessation of the drainage of pus. At this stage a two-minute treatment was given directly over the frontal sinus using 6½-in. spark-gap, 5 ma., 3 mm. of aluminum and 8-in. distance. For twelve hours the pain increased, and the following day the drainage tract was sponged out with adrenalin and cocain. The second day the discharge was less and the pain had disappeared. A day later another *x*-ray treatment was given for a minute and within the next five days all discharge stopped and has never returned. Up to this time the patient has had no further sinusitis. A re-examination in 1919, and another in July, 1922, show a greatly increased density of the sinus in question.

This permanent change will be discussed while considering the pathology of the condition.

Cases number X and XI will be given at this time, and short reports of the remaining cases with the lantern slides. These two cases appear to border on the chronic stage of sinusitis.

Case number X is an attorney. In January this year he had the flu. A persistent nasal discharge kept up until April when he consulted a nose and throat specialist. The intranasal examination revealed an infected left antrum, which was punctured and irrigated. During April, May and June the antrum was irrigated 25 times. On July 1st his physician went on a vacation. On July 5th the patient came to our office for examination and advice because of great pain and discomfort. The *x*-ray films revealed both frontals involved as well as the left antrum. He was given a two-minute treatment through the frontals from the left lateral position and the same treatment through the left antrum. Two days later he declared that all his discomfort and confusion had stopped within a half-hour after the treatment. The second treatment was then given directly over the anterior surface of the sinuses involved. Within a week all discharge had stopped and the patient said he felt better than at any time for six months, which dated back to the flu infection in January. He has continued to improve and up to this time has shown no further signs of sinusitis.

Case number XI is a manufacturer, aged 59. He has had for two years a constant watery discharge from the nose accompanied by frequent sneezing, due to an irritating dust in his manufacturing plant. He would require a dozen handkerchiefs during the day and nearly as many at night. In January, 1922, the discharge became yellow, and by April he began to have pain over the right eye and in the right antrum. He then went to a nose and throat specialist and only the antrum was treated. It was punctured and irrigated eight times during a period of five weeks, with no improvement. On July 11th he came in for examination. The *x*-ray films showed a marked cloudiness of the right antrum and right frontal sinus. An *x*-ray

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922.

treatment was given the same day and again in two days. He has required seven *x*-ray treatments and no further irrigations to clear up his symptoms. Estivin was used to help check the profuse secretions, which would begin as soon as he would read for a few minutes.

Now let us consider the etiology of acute purulent sinusitis. According to Ballenger, Phillips and Skillern, infection of the antrum may be due to:

1. Direct extension from the nasal mucosa, as in coryza.

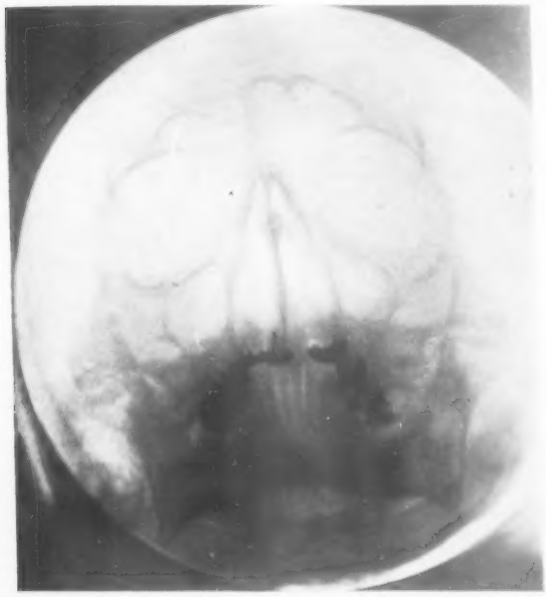


FIG. 1. Case I. July 31, 1922. Right frontal sinusitis.

2. Infectious diseases, particularly influenza.

3. From the alveolus. About 20 per cent of the cases of maxillary sinusitis are thought to originate from an infected tooth.

4. Through contamination from an overlying sinus, such as the frontal or anterior ethmoid cells.

5. Foreign bodies.

6. Traumatism.

7. Osteomyelitis, syphilis, tuberculosis and malignant tumors. All the cases in this series come under the first two headings, namely, coryza or influenza, which are the most frequent causes of sinusitis.

Infection of the frontal sinus occurs much the same as infection of the antrum, with two exceptions, namely, the contaminated overlying sinus, and the infected alveolus.

PATHOLOGY

"The first pathological changes that occur in acute purulent sinusitis are chiefly confined to the lining mucosa which becomes inflamed, swollen and edematous. In other words, only the superficial layers of the mucosa are involved in the acute purulent condition, while in the chronic disease all the layers of the mucosa undergo

FIG. 2. Case I. Aug. 23, 1916. Right frontal sinusitis.

pathological changes. Serum and leucocytes escape through the epithelial covering of the mucosa, where they mix with bacteria, epithelial debris and mucus. The secretions, at first thin and watery, become thick and tenacious and as the purulent stage progresses the leucocytes are thrown out in immense numbers. Unless the process is speedily arrested the tissue changes become permanent and a chronic sinusitis is established."

So much for the etiology and pathology. The question remains: What occurred when the *x*-ray was applied to the cases of purulent sinusitis to cause a complete cessation of all the symptoms and drainage of pus within three days to three weeks, where with drainage and irrigation no improve-

ment had taken place in one to three months?

Perhaps it is too early to say that the application of the *x*-ray cured the sinusitis. At least it seems fair to state that the application of the *x*-ray was coincident with the relief of the symptoms and the cessation of pus in all the cases. One case has remained well for six years. The other cases are well at this time and their condition will be examined from time to time and reported later.

FIG. 3. Case X. July 5, 1922. Both frontals and antrum.

The thought of the writer is that rapid or forced resolution of the inflammatory process takes place; the leucocytes no longer escape and even the secretion of mucus ceases for a time. The mucosa, however, does not return to normal, but remains thickened, as evidenced by Case I, where the cloudiness has remained for six years after the cure of the sinusitis.

Chronic suppurative sinusitis with granulations, polypi or necrosis of the bone is the end result of the acute condition which every method of treatment seeks to avoid. The *x*-ray offers a method of preventing the acute catarrhal condition from becoming purulent and the acute purulent sinusitis from becoming chronic.

Two warnings must be given in the application of the *x*-ray to a purulent sinusitis. First, the eyes must be protected from the ray, or dimness of vision may result. Second, the chronic condition should not be treated with the *x*-ray, for no result can be expected where polypi have formed or necrosis of bone is present. Again, if there is pent-up pus, septicemia may result.

CASE REPORTS

CASE II. Male. Jan. 21, 1922. Patient caught cold Dec. 27, 1921. All the upper air passages became inflamed and a frontal sinusitis developed eleven days later. Daily intranasal treatment of argyrol was given and free drainage was maintained by the aid of adrenalin and a mild cocain application. On account of a bronchitis the patient could not get to our office for an *x*-ray treatment for two weeks after the beginning of the sinus infection. After one treatment the pain and tenderness began to disappear. Within one week the discharge stopped. Incidentally the patient was cured of a chronic catarrhal condition, of which he had complained for a year.

FIG. 4. Case XI. July 11, 1922. Right frontal and right antrum.

CASE III. Female. Feb. 1, 1922. Intranasal examination revealed pus coming from the right antrum. The patient had had a yellowish nasal discharge for two months. The *x*-ray film revealed definite cloudiness of both frontals as well as the right antrum. Four treatments were given at four-day intervals. The discharge

lessened and finally disappeared in about four weeks.

CASE IV. Male. March 13, 1922. This patient came in for relief of pain over the eyes. The *x*-ray film showed an increased density of the left antrum. Intranasal examination revealed only a white mucous discharge. However, a one-minute treatment was given and prompt relief from pain followed within a half-hour. Pus appeared the next day and lasted only twenty-four hours. No further treatment was given. This case should be regarded as having received the *x*-ray treatment during the acute catarrhal stage.

CASE V. Female. March 17, 1922. The patient complained of pain over the left eye, of one week's duration. Intranasal examination revealed considerable pus in the left nostril. The *x*-ray film showed slight cloudiness of the left frontal and marked cloudiness of the left antrum. Two *x*-ray treatments were given, a week apart. All symptoms and discharge had disappeared at the end of two weeks. Four months later a re-examination was made. All trace of the frontal sinusitis had disappeared on the *x*-ray film. However, the cloudiness of the antrum had increased.

CASE VI. Female. March 21, 1922. Intranasal examination revealed pus pouring out over the left inferior turbinate. The *x*-ray film showed that only the left antrum was involved. Only two *x*-ray treatments were given this patient. All symptoms and the discharge disappeared within ten days. This patient had had symptoms of a sinusitis for only two weeks.

CASE VII. Male. March 28, 1922. The patient complained of pain in the region of the left antrum following an acute coryza. Intranasal examination revealed pus coming from the antrum. The usual intranasal applications and anodynes were given for two days. As soon as the patient could come to the office, the *x*-ray examination was made and the left antrum was found to be the only sinus involved. Two *x*-ray treatments were given, three days apart, with relief of all symptoms and the discharge within one week.

CASE VIII. Male. April 4, 1922. This patient had been ill for four months as the result of the flu in December, 1921. After his pneumonia cleared up in January he complained of pain in both antra and a greatly increased nasal discharge. *X*-ray films made in April showed marked infection of both the right and the left antrum. This patient received two *x*-ray treatments a week apart, all his nasal discharge disappeared within two weeks from the first treatment, and he returned to work a week later. No irrigations had been given either antrum. In the writer's opinion, this case would soon have resulted in a chronic sinusitis.

CASE IX. Female. June 8, 1922. The patient had the flu in January, 1922, and gave a history of repeated "colds in the head" for five months. About June 1st a severe pain began over the right eye and in the right cheek and temple. On June 8, 1922, the *x*-ray films revealed a marked cloudiness of the right antrum. An *x*-ray treatment was given the same day and repeated three days later. Two other treatments were given at weekly intervals. It took about three weeks from the first treatment for all the discharge to disappear.

CASE XII. Female. July 24, 1922. The patient caught cold about July 10th, and in less than ten days began to complain of severe pain over the left eye. Upon intranasal examination, July 24th, free pus was seen coming down from the left frontal. Roentgenograms were made and definite cloudiness was seen in both frontals, left ethmoid and left antrum. A two-minute treatment was given the frontal sinus from the lateral position. Two days later it was treated anteriorly. The left antrum was also treated at this time. Marked relief was given by the first treatment, for twelve hours, and complete relief by the second treatment. All drainage ceased within five days.

# THE ROENTGEN THERAPY OF TINNITUS AURIUM*

BY LYELL CARY KINNEY, M.D.

SAN DIEGO, CALIFORNIA

TINNITUS is the most distressing symptom in ear pathology. It is the chief complaint of the ear cases referred for roentgen therapy, and the control of this symptom is the index of success or failure.

Serious roentgen therapy of ear conditions during the past seventeen years in the European clinics has not yielded encouraging results. The first work in *x*-ray therapy is by Joulin in 1908, who reported 10 cases of otosclerosis treated by small weekly doses of *x*-ray, 6 of which showed improvement of the tinnitus and some improvement in voice perception, but there was no improvement to objective tests. Ortloff, in 1913, reported 10 cases in which he found that the slight improvement was invariably followed by a return of the tinnitus and deafness. Siebenman, of Bale, has experimented for nine years with roentgen therapy in otosclerosis and concludes: "The results are only to a small degree positively encouraging."

Two years ago, the subject was revived by Dr. C. F. Stokes who reported astonishingly good results from "electronizing the pituitary auditory region" with minute doses of *x*-rays.

Exaggerated rumors of such results have come to the West and our otologists are asking for the cooperation of the roentgenologist in the treatment of tinnitus aurium.

Persistent tinnitus is usually the symptom of organic pathology. It may come as the result of a neuritis of the eighth cranial nerve, tuberculosis, suppurative otitis media or disease of the labyrinth. Most frequently it is a symptom of otosclerosis or chronic otitis media, and the treatment of tinnitus means an attack upon one of these two types of pathology.

The cause of otosclerosis has been assigned to the whole list of faulty metabolism, heredity and infection. It is probably not of endocrine origin, for the disease occurs in healthy individuals who show no other symptoms of glandular pathology. On the other hand, patients suffering from glandular deficiencies such as acromegaly, Addison's disease or hypothyroidism show no tendency toward otosclerosis. Whatever the cause, the pathology of otosclerosis consists in changes in the bony capsule of the internal ear. There is absorption of the normal bone in front of the oval window which starts around the capillaries of the Haversian canals accompanied by increased vascularity and overgrowth of the cartilage cells. The absorption is followed by fibrosis or the deposit of a typical loosely trabeculated bone. Exostoses and bone deposits occur around the foot of the stapes, resulting in a true ankylosis. Later there are areas of osteitis throughout the bony capsule of the cochlea, followed by nerve degeneration. Whether these bony changes are trophic or inflammatory, they closely resemble the bone pathology in hypertrophic osteoarthritis. The progress of the disease is insidious and the symptoms of tinnitus and deafening are rarely noticed until there are advanced bony deposits around the articulation of the stapes.

With this pathology in mind there are only two things that can be expected from radiation: control of the capillary congestion and checking of the cartilage-cell proliferation. In other words, the most that can be accomplished is arrest in that stage of bony deformity to which the case has arrived. Thus any treatment of the tinnitus resulting from otosclerosis offers very little.

I have had one case where *x*-ray treatment has relieved the tinnitus and there has been improvement in the deafening. My other cases were absolutely not influenced by *x*-ray. Fortunately, otosclerosis is rare.

Tinnitus is usually a symptom of a chronic inflammation of the middle ear and Eustachian tube with the source of pathology in the nasal pharynx.

The lower half of the Eustachian tube is surrounded by a cylindrical layer of adenoid tissue and the mucosa of the pharyn-

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922.

geal orifice is padded with cushions of adenoid cells. This adenoid tissue is subject to the same infection and hypertrophy as that in the pharynx, and such infection will result in a mechanical obstruction within the tube. The outlet of the tube may be obstructed by adenoids or polypoid turbinates. Any obstruction or inflammation in the pharynx will result in altered air tension and infection in the middle ear with the resulting symptoms of tinnitus and deafening.

The treatment of this type of tinnitus and deafness consists in the removal of the pathology in the pharynx and the restoring of normal aeration and drainage to the middle ear. It has been proven that the *x*-ray will play a definite part in removing certain of these causes. The adenoid tissue in the pharynx can be controlled by *x*-ray. The swollen adenoid layer of the Eustachian tube and the adenoid collar at its outlet can be eliminated by *x*-ray. The latent infection in the middle ear and pharynx can, in some cases, be destroyed. Capillary engorgement and early fibrosis in the middle ear can be lessened. All these are factors influencing tension, irritation and fixation in the middle ear. It is therefore logical to expect improvement in chronic otitis media and the resulting tinnitus from *x*-ray treatments. But here, as in otosclerosis, no change can be expected in terminal pathology, such as dense adhesions, bony changes in the ossicles or resulting degeneration of the labyrinth.

Realizing that any permanent relief must come from changes in pathological anatomy and not from endocrine stimulation, I have given a reasonable quantity of radiation. The dose has been small, but calculated to deliver approximately one-half of an erythema to both ears in one month. I have used 135 kv., 5 ma., filtered through 6.0 mm. of aluminum at 60.0 cm. distance through a portal 5.0 cm. in diameter. The exposures have been two minutes on each side daily for eight doses with a weekly interval of rest. The average case receives three such series, when, if there is no relief, the treatment is abandoned. If there is appreciable improvement, treatment is continued at monthly intervals.

Practically all the patients complained of increased tinnitus and rhinitis, and some of dizziness for the first three or four treatments. If there is going to be an improvement in the tinnitus, it will usually begin at the end of the first week.

This technique has been followed through a series of 14 cases of tinnitus from chronic otitis media, because of definite requests from our otologists for "minute frequent doses." In 40 per cent of these cases, we have found permanent decrease in the tinnitus and marked improvement in the hearing to objective tests.

In conclusion: The suffering from tinnitus is most distressing and some cases obtain definite relief from *x*-ray therapy. When tinnitus is the symptom of otosclerosis, the most that can be hoped for is to prevent its increase, and then only in the early cases. When tinnitus is a symptom of chronic otitis media, improvement may be expected from roentgen therapy in some cases. Such improvement is due to the known anatomical changes produced by the *x*-ray, and there is no reason to attribute it to endocrine stimulation.

The *x*-ray should be considered only as one of the necessary factors in clearing up the infection and obstruction in the pharynx, Eustachian tube and middle ear.

I want to thank Dr. Andrew Wessels of San Diego for his earnest cooperation in this work.

BIBLIOGRAPHY

1. Gray. Otosclerosis. *Laryngoscope*, xxxi, no. 7.
2. Hays. Modern conception of deafness. *Laryngoscope*, xxxi, no. 9.
3. Webster, J. H. *X*-Ray Treatment of two cases of otosclerosis. *Arch. Radiol.*, xxvi, 69.
4. Jaulin. Traitment de l'otite scléreuse par les rayons X. *Arch. d'électric. méd.*, 1908, p. 92.
5. Ortloff. Über therapeutische Versuche mit Roentgenstrahlen bei der Otosclerose. *Arch. f. Ohrenh.*, 1912, xc, 233.
6. Stokes, Chas. F. Discussion. *Laryngoscope*, xxxi, 739.

# DISCUSSION ON PAPERS OF DRS. McCOY, OSMOND AND KINNEY*

DR. HICKEY. The reading of medical history is both interesting and instructive. From it one gains the ideas of different types of treatment which come and last for a short time and then fade away. The lesson to be learned is that new lines of treatment should always be viewed conservatively.

I hope Dr. Osmond will pardon me if I discuss his paper frankly. Personally it does not seem to me to prove the point which he endeavors to bring out. For many years I was engaged in nose and throat work and one of the things that I well remember is the remarkable way in which many cases of sinus trouble would get well without any very special line of treatment. A great majority of the cases of acute sinusitis tended to spontaneous recovery.

In considering the *x*-ray treatment of sinusitis we feel that we must approach the subject with a good deal of conservatism in view of what we know about natural tendency to get well through the curative powers of nature. The exceptions to this rule are where the infection comes from the alveolar process. While the report of Dr. Osmond is very encouraging and stimulates us to further observation, still I do not feel that we should go away from the meeting with the idea that his paper is any more than a preliminary report.

In regard to the treatment of defective hearing, I am very glad to state that the paper of Dr. Kinney has crystallized some ideas on the question which is so popular at the present time. The treatment of defective hearing with *x*-ray seems to be one of the favorite indoor sports of roentgenologists at the present time. It must be confessed, however, that it is very difficult to classify the results we are getting, inasmuch as we are dealing with a class of patients who are very anxious to recover and who watch their own symptoms very minutely. When the president asked me to discuss this paper, I had treated at the time some three or four cases with the so-called Stokes method. I had obtained no definite results. I then went over to the Otologic Department of the University of Michigan and asked them to send over some of their cases of defective hearing. We treated a number of cases according to the advised technique and found when they were re-examined by the Otologic Department that there was no benefit derived. An interesting feature, however, was that certain of the patients seemed to think they were some better.

An interesting observation was that we placed some plates against the head at the time of treatment and found that the amount of ray which went through the skull was scarcely enough to produce any perceptible shadow upon the plates. Certainly the quantity of ray which the intracranial tissues received was very small.

The type of treatment which Dr. Kinney describes is, we think, quite rational and marks a distinct advance in the treatment of defective hearing.

DR. LAW. I do not know why I am to discuss this, because as you all know, I fight shy of treatments. The only thing I can say is regarding this Stokes treatment.

I heard about Stokes treatment and was asked to go up and investigate it. I know very little about dosage except what I have heard. It hardly seems possible to me that a 4-in. gap, 30-in. distance, 5 ma. for 20 odd seconds would do much good. However, it was worth the trial, because certainly the *x*-ray does queer things, and no doubt there are things we know nothing about so far. We cannot condemn any use of the *x*-ray until it is thoroughly investigated.

Dr. Stokes gave us the privilege of sending in a number of cases at any time when we cared to. Cases were sent up there and strangely, every one of those patients said they heard better, that is, the sounds seemed clearer. Then they were tested, as they are always tested, and with the exception of the voice test there was no difference. It is pretty difficult to make the voice give the same density twice in succession, especially when you are anxious that the patient should hear better. Then we tested them under the best known scientific methods, sent them up for treatment, and re-tested them without any change in the ultimate results. That was not quite sufficient, so the said cases were treated elsewhere, and there was no change, hence, there may be a certain psychological element there. However, we should not condemn the thing until you, who understand those things and understand the technique, have thoroughly investigated it. Personally, I am not in a position to do it.

Dr. Kinney's work is well worth thorough investigation. I am sure that when I get back to New York, we will see what we can do with

* Papers read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922. That of Dr. McCoy appeared in March, and those of Drs. Osmond and Kinney precede this discussion.

some of these obstinate cases. As Dr. Hickey said, all acute cases have a tendency to recover for a few days at a time.

I am sorry Dr. Osmond showed plates which in the second examination were not the same as in the first. In none of them did I notice a tendency to much illumination. It is well worth trial because we know that in many cases going for *x*-ray examination of the sinuses or mastoid, the patient is relieved and sometimes the discharge increases for a time and suddenly decreases. Now we know there is an electrostatic field encircling the patient and this does help so far as certain things are concerned. All these things are new and I think instead of condemning them we should try them, but with hundreds of cases rather than with a few cases.

Dr. Remer. It seems that the natural tendency in a new field is condemnation. While I do believe, on the other hand, that it requires a certain amount of definite and long-continued work to bring out definite ideas and results, at the same time, I do not think we should be too prompt to condemn.

I do feel in regard to Dr. Stokes' work, that the amount of rays used, the way in which they were used, and the amount of dosage obtained could scarcely result in any advantage. In Dr. Kinney's work there is, as Dr. Law said, a definite pathological condition. Further, I believe that in our work the cases must be selected to obtain results: then where you have an active inflammatory pathological condition, with a sub-acute inflammatory condition, you are apt to get results. The *x*-ray will have no influence on a negative result. In most of the cases of tonsils treated, you find adenoid tissue and lymphoid tissue. You also find a certain amount of inflammatory condition surrounding the Eustachian tubes. Dr. Kinney states that he uses the method which is recognized. In that way you do get a definite result of the pathological condition surrounding the Eustachian tubes. Where there is that condition you undoubtedly have tinnitus. By relieving the condition, you open up the pocket, and in that way, get relief of the tinnitus. Your end results are not obtained by changing the anatomical condition, but by changing the pathological condition.

In regard to treatment of sinuses I have had no experience, and with Dr. Law, I am rather inclined to be skeptical; yet I do not think we should be too quick to condemn. However, we must have a larger number of cases than have been reported to accept it.

Dr. Stewart. Last winter I had the privilege of hearing Dr. Stokes, and as I had not had any experience, I could not say much except with reference to the dose indicated. I said that I did not believe a sufficient amount of *x*-ray was given possibly to benefit these patients, and I thought that probably the effect was entirely psychological. Stokes' response was that a great many had stated the same thing, but he considered 2,000 cases sufficient to overcome that argument on psychology.

Dr. Lewald. I have some definite evidence along this line, i.e., a more delicate instrument for testing the hearing has been evolved in the last few years—the Fowler Audiometer. This gives much more accurate intensity and frequency measurement than any instrument heretofore devised.

Dr. Edmund P. Fowler, otologist, has tested ears before and after treatment by Stokes, and he told me very definitely that he could detect no improvement in the hearing of patients, although Stokes was reported to have assured them they were greatly benefited or cured of their deafness, and the patients themselves temporarily imagined this to be true.

Dr. Kinney (closing discussion). Last year Dr. Webster published a description of two cases of otosclerosis that he believed had been arrested. Immediately Dr. Barclay called him to task for it, maintaining that it was untimely and aroused unwarranted hopes. I thought the same thing when we began to hear about the treatment of ear conditions from the East. We are approaching our cases in just that way, trying to discourage any psychic element. We tell them we do not expect any relief from the deafness but will try to help their head noises.

I do not want you to go away from this meeting thinking that I am suggesting anything else than helping the tinnitus in some cases, and in these cases attacking a definite pathological condition. Of course the results in a small series prove nothing. This series was presented simply to start a discussion to see what you gentlemen were thinking along this line. It is an effort to obtain a rational basis for the relief in some of these people who are improving. I do not believe that the relief comes from electronizing the pituitary, but that what improvement occurs comes from known demonstrable changes in pathological anatomy.

Dr. Osmond (closing discussion). My purpose in presenting this paper before the Society is to get you to use this method in some sinus cases. My series is not large; however, I am firmly convinced of the value in this method of treatment. If each one of you will use the method on a small series of cases, a more convincing conclusion for you can be formed

from the total number. Choose your cases. I realize that a great many acute sinuses will get well of their own accord. On the other hand, we know that some do not. If we can prevent an acute sinus from becoming chronic, it is worth while to do it.

While receiving my post-graduate instruction in nose, ear and throat work, I was impressed by the difficulty encountered in the treatment of chronic sinusitis of the accessory air sinuses and by the remark of Dr. A. J. Houston of San Francisco at that time: "What in the world can be done to stop these chronic sinusitis cases? I have been searching in Europe and this country in hope of finding something more satisfactory in the treatment of this condition."

It is a difficult thing to cure chronic sinusitis; therefore, I believe that we should do our part to help to prevent a condition from becoming chronic. I do not think there is much value in treating chronic sinusitis with the *x*-ray. If any of you try it, be sure to make an intranasal examination first to see if pus is there, and to see if it is draining. If there is not sufficient drainage, you might do harm.

In regard to the two positions, the following method was used: on every sinus case two posteroanterior exposures were made, and one lateral. The posteroanterior position consisted of one made in the usual 23-degree angle and the other in the Waters position. If either position ruled out sinusitis, the case was not used in the series. If both positions showed sinusitis, the film was chosen for exhibit which most clearly outlined the condition.

In closing, let me say that if any of you have ever had sinusitis and have had it treated by irrigation, you would appreciate by contrast the absence of discomfort afforded by *x*-ray therapy.

# THE VALUE OF THE ROENTGEN STUDY OF MASTOID DISEASE IN CHILDREN UNDER FIVE*

BY WM. A. EVANS, M.D.

DETROIT, MICHIGAN

THE practice of general roentgenology affords one an excellent opportunity for the study of anatomy in all of its variations. Early in my work in this field, my observations of mastoid structure in infants and young children were contrary to the usual teachings and writings. As my experience with clinicians broadened, and I was called upon to interpret abnormal mastoid structure in clinical terms, I became convinced that those who denied the value of the roentgen diagnosis in mastoid disease in early life probably spoke from inexperience or misinformation. So, when it was stated in the very excellent work on "Mastoids"[1] by the leading authority in this branch of our work that "Except in very unusual cases the mastoid process does not show cellular development before three years of age, and usually not until about the fifth year," it seemed advisable to publish our observations and experiences, since they were not in accord with the generally accepted ideas.

Recent publications in English by anatomists on the structure of the mastoid process agree with the older works as to the tardy pneumatization of the mastoid.

Goldstein,[2] in 1921, stated: "The mastoid process contains no air cells at birth, but toward puberty the process becomes pneumatic."

Bigelow,[3] of Providence, through his knowledge of the work of Cheadle, was familiar with the early formation of pneumatic cells, but was impressed with the frequency of the persistence of the infantile type to adult life, but the frequent development of the adult type of mastoid structure in early childhood was not noted.

Professor E. Zuckerkandl,[4] the leading European anatomist on the temporal bone, also observed the early formation of pneumatic structure, and states that in the second year cellulae can be present, and in the third year the process resembles frequently the process in the adult. It will be instructive to quote verbatim from the above article as to the embryology of the mastoid:

"Mastoid process of the child, the pars mastoidea, is already present in the

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922. Discussion of this paper and the others in the same symposium will appear in a later number of the JOURNAL.

embryo; a cone-like, pneumatic process develops, only very much later. Of the pneumatic spaces, first the antrum appears, which Schwartze and Eysel have observed already in the five-months' embryo. In the newborn there is a little knob at the spot of the mastoid process, the anterior plane of which is taken up by a triangular process of the squama (lamina mastoidea squamae). Between both there is to be found the fissura mastoidea which passes away only after the complete second year and which contains a connective tissue, continuation of the outer periosteum. Not infrequently, portions of the same or the whole suture persist. This is important to consider, on account of its relation to the propagation of diseased processes from outward inwardly, and vice versa. The formation of the mastoid cells begins at the posterior end of the mastoid antrum, and continues at first horizontally toward the sigmoid sinus, so that the upper portion of the pars mastoidea becomes pneumatic sooner than the mastoid process. The cell formation in the same also starts through the antrum which pushes a prolongation toward the tuberculum mastoideum. This evacuation grows in the course of the first month. At the end of the first year the tuberculum contains already a comparatively large cavity and becomes cone-shaped. In the second year cellulae can be present, and in the third year the process resembles frequently the process in the adult."

Very little reference is made to the value of *x*-ray diagnosis in the literature on the clinical aspect of mastoid disease.

In Philip D. Kerrison's book,[5] "Diseases of the Ear," published in 1921, no mention is made of this method of diagnosis.

Amberg,[6] has an article in volume xxvi of *The Laryngoscope*, in which he recognizes the value of the aid given by the *x*-ray method of study, but his cases did not include a patient under five years.

Bigelow,[3] mentions the failure of twenty-one out of twenty-five otologists to recognize the value of the *x*-ray examination in acute infections of the middle ear and its complications.

Articles in the roentgen literature on this subject are few. Sidney Lange,[7] in 1909, made the following statement:

"Upon the mastoid skiagrams of children under ten years of age the mastoid appears entirely spongy or diploetic, there are no visible cells and the tip is undeveloped. The tip at this time consists simply of an inner and an outer table, with little cancellous bone between, and shows no structure upon the skiagram. From ten to fifteen years the mastoid takes on the pneumatic characteristics, large cells appear and grow downward toward the tip, its diploetic structure finally giving way to the more or less pneumatic adult type." No doubt his later experience has proven that the above statement is not in accordance with the facts.

Dr. Wm. H. Stewart,[8] of New York, as early as 1913, in an article entitled "Radiographic Findings Illustrating the Anatomic Development of the Mastoid Bone," states that examinations of normal children as to mastoid structure demonstrated that distinct and well-formed pneumatic cells occur as early as two years, and Gerber,[9] in collaboration with Bigelow, agrees with Stewart as to the early presence of pneumatic cells.

The material for this paper was obtained from Harper Hospital and our office records for the first six months of the year 1922. In that period 254 cases were referred for mastoid examination. Classified according to age, we find that 7 were less than one year of age; 46, less than five years of age; 52, from five to ten years of age; and 147, over ten years of age. Of the 53 cases of less than five years, 39 were operated on and operative results were available. We were able to classify, anatomically, all of the proven cases according to the classification of Cheadle, which arrangement was found acceptable by Gerber and Bigelow. That is, three types of infantile mastoid: First, the diploetic form; second, the dense form; third, the pneumatic infantile; and finally, the adult type. Of the 39 cases, 6 were classified as the diploetic infantile; 1 as the dense infantile; 13 as the pneumatic infantile; and 19 the adult type.

These figures indicate that pneumatization of the mastoid structure occurs much earlier than all the former writings indicate, and that the adult structure is

found in a much larger proportion than was formerly believed.

We further classified the types in our operative cases as to years. Of the patients less than one year of age, 5 presented the pneumatic type, and only 1 the diploetic type. In the second year we found 1 of the diploetic infantile; 1 of the dense infantile; 3 of the pneumatic infantile; and 3 of the adult type. In the third year, 2 presented the diploetic type, 1 the pneumatic infantile, and 5 the adult type. In the fourth year, 2 were of the diploetic infantile type, 2 of the pneumatic infantile, and 7 of the adult. In the fifth year, we found 1 of the pneumatic infantile, and 5 of the adult structure.

TYPES OF MASTOID STRUCTURE

| Age | 1 | 2 | 3 | 4 | 5 | Total |
|---|---|---|---|---|---|---|
| Diploetic. | 1 | 1 | 2 | 2 | | 6 |
| Dense | | 1 | | | | 1 |
| Pneumatic infantile | 5 | 3 | 1 | 2 | 1 | 12 |
| Adult | | 3 | 5 | 7 | 5 | 20 |
| Total | 6 | 8 | 8 | 11 | 6 | 39 |

Since the above conditions exist, stereoscopic plates of the mastoid regions of children will give information comparable to that of adults, and in all cases of complicated middle-ear infection, plates should be obtained, so that the surgeon can know in advance the area he is to explore, especially as regards type and extent of structure, and direction that the infection probably took.

Even in those cases in which there is no cellular structure, that is, where the structure is diploetic or dense, we believe that valuable information can be obtained from stereoscopic plates. It is not possible to be as exact in the classification of the degrees of bone change, but in cases where there is definite disturbance of bone structure, the lesion can be usually demonstrated.

In the interpretation of the mastoid plates as regards disease and clinical indications, we have followed the classification of the changes adopted by Dr. Lange:[10] that is, the mild or first degree mastoiditis in which there has been a replacement of the air content with exudate but no change in the cell walls; the second degree, in which is noted in addition to the absence of air content, a change in the bone structure, the cell walls being indistinct and hazy, and in places possibly a coalescence of a number of the cells; the third degree, in which practically all of the cell structure has broken down. First degree mastoids are not considered surgical, but the second degree is usually surgical, and the third degree, always surgical.

In all the cases studied in which the degree of pathology and surgical indications were stated, the findings at operation coincided with the pathology described by the roentgenologist, with the exception that in several cases the destructive process had gone beyond that suggested by the roentgenograms. In two cases the indications for treatment, as suggested by the roentgenograms, were disregarded by the operator, and a second operation was necessary: that is, the surgeons considered a drainage of the antrum sufficient when there was roentgen evidence of pneumatic structure which was infected, and it was later necessary to re-operate and remove the infected cellular structure.

Continuing the quotation from Law's book on "Mastoids:"[1] "This fact (referring to the absence of cellular development under the fifth year), together with the difficulty in immobilizing the head of an infant—" We agree with the technical difficulties and have found it necessary in practically every case to give an anesthetic, but under these conditions very satisfactory plates have been obtained. It is important, of course, that the plates be stereoscopic, and that the technique used be one that shows to best advantage the mastoid area. Our custom is to place the patient in the prone position, with the head turned first to the right and then to the left on an angle board; the head is fixed by a mechanical device and further held by an assistant. The central rays for the first

plate are directly vertical, and for the second plate the shift is made backward and the central rays angled anteriorly.

Of late we have been using the so-called "flash" method, to obviate the necessity of a general anesthetic in obtaining plates. The technique of this involves the use of double-coated films and screens; the voltage varies from 55 to 70,000; about 30 milliamperes, and about $\frac{1}{10}$ to $\frac{1}{30}$ of a second, depending upon the age of the child and the size of the head.

### CONCLUSIONS

1. Pneumatic mastoid structure is frequently observed before the end of the first year of life.
2. The adult mastoid structure can be observed as early as the second year.
3. Stereo plates of good quality, of children under five, have definite diagnostic and prognostic value.

### BIBLIOGRAPHY


1. Law, Frederick S. Mastoids: Annals of Roentgenology. Paul B. Hoeber, New York, 1920.
2. Goldstein, Joseph. *Ann. Otol., Rhinol. & Laryngol.*, June, 1921.
3. Bigelow, F. Nolton. Types of mastoid structure with special reference to their recognition by means of stereoradiography. *Ann. Otol., Rhinol. & Laryngol.*, 1918, xxvii, 54.
4. Zuckerkandl, Prof. E. *Handb. d. Obrenbeilk.*, vol. i, pp. 33, 34.
5. Kerrison, Philip D. Diseases of the Ear. Philadelphia, 1921.
6. Amberg, Emil. Some remarks on the *x*-ray diagnosis of mastoiditis. *Laryngoscope*, 1916, xxvi.
7. Lange, Sidney. The *x*-ray examination of the mastoid region. *Am. Q. Roentgenol.*, Dec., 1909, ii, no. 1.
8. Stewart, Wm. H. Radiographic findings illustrating the anatomic development of the mastoid bone. *Ann. Otol., Rhinol. & Laryngol.*, 1913, xxii, 677.
9. Gerber, Isaac. Some observations of the mastoid structure as revealed by roentgen ray examinations. Am. J. Roentgenol., Jan., 1919, p.1.
10. Lange, Sidney. Practical results in *x*-ray examination of the mastoid. Am. J. Roentgenol., June, 1914, i, no. 8.


# A REVIEW OF THE TREATMENT OF HYPERTHYROIDISM BY ALL METHODS, WITH A SUMMARY OF THE AUTHORS' EXPERIENCE WITH ROENTGEN THERAPY*

BY THOS. A. GROOVER, M.D., A. C. CHRISTIE, M.D., AND E. A. MERRITT, M.D.

THE rational treatment of hyperthyroidism had its origin in the thyreogenic theory first clearly set forth by Moebius in the year 1886. Exactly a century before that time, Parry had recognized typical cases of exophthalmic goiter, and during the intervening century many noted names were connected with the study of the disease, notably: Graves, Basedow, Stokes, Charcot, and Trousseau.

The foundation of all subsequent work was laid by Moebius who brought forth the theory that "Basedow's disease is an intoxication due to the morbid function of the thyroid gland" and who stated that the disease can be looked upon as a "hyperthyroidization." Clinical observation and experimental work since that time have served to establish thoroughly the correctness of his views. The term "hyperthyroidism" now has a sound scientific basis. It serves to designate a syndrome characterized by an increased metabolic rate and by certain well-known clinical manifestations.

Kendall's work in 1914, in separating the pure chemical compound, thyroxin, from the thyroid gland, his subsequent work in proving that thyroxin is the active principle of the thyroid, and finally his demonstration of the chemical composition of this active principle, not only added greatly to our knowledge of diseases of the thyroid gland, but is one of the most complete and beautiful pieces of work in the entire history of medical science.

The investigations of Du Bois, of Plummer and his associates, Boothby and Sandiford, and of Means and Aub, have established beyond reasonable doubt that the basal metabolic rate is an accurate

* Read at the Midwinter Meeting of the Eastern Section of The American Roentgen Ray Society, Atlantic City, N. J., Jan. 25-27, 1923.

diagnostic measure for the recognition of the presence or absence of hyperthyroidism. Plummer has clarified the problem by showing conclusively that there are two types of hyperthyroidism: true exophthalmic goiter and adenoma with hyperthyroidism. The two conditions differ not only in their underlying pathological anatomy, their mode of onset, their clinical course and their prognosis, but also in their treatment.

It is not our intention to attempt to recount in this paper the different methods that have been used in the treatment of hyperthyroidism. Their name is legion. The use of iodine and the iodides internally, and of iodine or iodoform for injection into the gland; the administration of thyroid or thymus gland preparations, of sera from thyroidectomized dogs or sheep; the milk of thyroidectomized goats; and the serum of Rogers and Beebe, have all been used and now generally abandoned. Today only two methods are in general use: surgery and roentgen-ray therapy. We wish to discuss the subject under the headings of (1) general management, (2) surgical treatment, (3) roentgen-ray treatment.

The general management of patients with hyperthyroidism is of major importance, whether the ultimate treatment is to be surgery or the roentgen-rays. It should be as well understood and as carefully carried out by the roentgenologist as it is by the surgeon or internist. We venture to recall here the details of the general management of this disease only because we believe that many who are called upon to treat patients with hyperthyroidism are lax in this very essential part of its treatment.

*Rest* is the first indication, and whether it is to be absolute or relative depends upon the severity of the disease. In severe cases a rigid regime of rest in bed and passive exercise must be carried out. The details of such a regime cannot be described here. In cases with mild symptoms, definite directions must be given as to amount of exercise, the necessity for long hours of sleep, and the avoidance of all unnecessary activity and of working to the point of fatigue. Rest to the mind is equally as important as rest to the body. Mental stress and worry must be avoided so far as possible. The physician must use every means in his power to gain the patient's confidence, to tranquilize her mind, and to create an atmosphere of optimism.

*The diet* must be regulated with care. Overfeeding is to be avoided, but in a disease characterized by increased catabolism, it is essential for the patient to have a large amount of food of the most nourishing kind possible. Much care should be used to make the food tempting to the appetite and at the same time of a kind that is easily assimilable and of high caloric value. It is equally important that the intake of fluid be as great as possible in order to assist in the elimination of waste matter produced by the increased metabolism. Hertzler recommends lemonade for this purpose, and to increase the caloric value of the food he adds about 50 gm. of lactose to each glassful.

*Cardiac symptoms*, if marked, may need special treatment. Tachycardia and palpitation usually subside when the patient is put at rest. If this does not occur, an ice-bag placed over the heart is often beneficial.

Digitalis is of great value in decompensation and fibrillation of the heart, but should not be used routinely because of its tendency to cause gastric irritation.

*The nervous symptoms and insomnia* will be greatly decreased by such general hygienic measures as rest, proper diet, and fresh air in the sleeping room. If insomnia persists, a full dose of sodium bromide at bedtime may be effective. Administration of opiates is very rarely justifiable in this disease.

*Gastric or intestinal complications* are usually controlled by careful attention to the diet; but if crises occur, lavage of the stomach and colon and mild cathartics may be necessary.

An essential element in the general management of hyperthyroid cases is the elimination of all sources of focal infection. The number of cases arising after influenza and tonsillitis strongly suggests the possibility of infection as a primary cause of the disease. Special

attention should be directed to the nasal accessory sinuses, the tonsils, and the teeth, and to the possibility of the presence of a chronically infected gall-bladder or appendix.

*Surgical Treatment.* The history of the surgical treatment of goiter is essentially the history of the art of general surgery. Some of the greatest names in surgery are connected with the operation for the extirpation of goiter. Until about 1890 all the important advances had been made by Swiss, German and Austrian surgeons. The most prominent among them were Theodor Kocher and Mikulicz. Tillaux in 1880 and 1881 operated on two patients with hyperthyroidism and Rehn operated for exophthalmic goiter in 1884. The work of Halsted in this country is too well known to need comment.

The good results obtained by surgical treatment established it, early in this century, as the treatment of choice for hyperthyroidism. During the past ten years the technique of operation and the surgical management have been so perfected by the work of C. H. Mayo, Crile, and others, that the mortality from operation has been greatly reduced and the end-results much improved. The generally accepted surgical treatment of hyperthyroidism may be given by summarizing a description by Pemberton[1] of the method of treatment carried out at the Mayo Clinic. The "patients with exophthalmic goiter fall into one of three groups: (1) patients on whom a primary thyroidectomy can be performed with reasonable safety, (2) patients concerning whom the wisdom of advising thyroidectomy is doubtful, (3) patients in whom indications for extended observation or preliminary measures are clearly defined."

In the first group are those patients whose basal metabolic rate is usually not above plus 55 per cent, in whom the severity of the disease is not increasing, and who have suffered no great loss of strength or weight. The second group comprises those whose basal metabolic rates are only a little higher than plus 55 per cent, but about whom doubt exists, possibly because of recent loss in weight, marked loss of strength, presence of slight edema, or undue apprehension on the part of the patient. A preliminary ligation of the superior thyroid is done on patients of this group and if no undue reaction occurs, it is followed by thyroidectomy in from seven to ten days. It is sometimes necessary to do a second ligation before the resection if there is marked reaction after the first. Group three comprises patients who must be kept under extended observation and who may need much preliminary treatment before a resection is advisable. The indications for this are a high metabolic rate, plus 70 per cent or higher, actual crisis or recent progression of the disease, extreme apprehension, marked weakness, loss of weight, chronic visceral changes, cardiac dilatation and chronic infection. Patients in this group are put at rest and carefully observed until the simplest surgical procedure, such as injection of quinin-urea or boiling water, may be done. This may be followed by successive ligations of the superior thyroid vessels, the interval between operations depending upon the degree of reaction. In most cases the ligations produce such improvement that thyroidectomy may be done safely.

The general management of patients with hyperfunctioning adenomatous goiter is the same as for those with exophthalmic goiter; but preliminary ligations, etc., are rarely necessary in these patients.

Pemberton gives the following statistics for the Mayo Clinic from July 1, 1920, to July 1, 1921. "Of the 281 patients with hyperfunctioning adenomatous goiter, 4 died, a mortality of 1.4 per cent. Of 677 patients with exophthalmic goiter, on whom 1,224 operations were performed (ligations and thyroidectomies), 23 died, a mortality of 1.87 per cent by operations and 3.39 per cent by patients."

The following is Crile's statement of his mortality: "Since the adoption of the method described in this volume, we have performed 1,783 thyroidectomies, including 1,022 thyroidectomies for exophthalmic goiter, with 25 deaths, a mortality rate of 1.4 per cent; and 783 ligations with 6 deaths, a mortality rate of 0.76 per cent."[2] It is impossible from this statement to determine the mortality rate in the patients with hyperthyroidism,

since no separate figures are given for the mortality in either exophthalmic goiter or toxic adenoma. It is fair to assume that the mortality rate in the 1,022 cases of exophthalmic goiter was greater than that in the 1,783 cases which included simple goiter. The 6 deaths following ligations might also be fairly charged to the surgical treatment of exophthalmic goiter.

Examination of the available statistics of operative mortality leads us to the conclusion that in the hands of the most skillful surgeons, with every facility for preoperative and postoperative care, the operative mortality in patients with exophthalmic goiter is somewhere between 2 and 4 per cent, and in hyperfunctioning adenoma about 1.5 per cent. The operative mortality of the average good surgeon throughout the world is undoubtedly considerably higher than this.

The end-results of surgical treatment in the best hands can be judged from statistics compiled by Judd from cases operated upon at the Mayo Clinic,[3] comprising 100 cases of exophthalmic goiter operated upon in 1914, and 100 cases of toxic adenoma operated upon in 1917 and 1918. The average elapsed time since the first series was six years, and since the second series was two years. The exophthalmic goiter cases showed 65.8 per cent cured, 13.6 per cent markedly improved, 5.6 per cent slightly improved, and 15 per cent dead from all causes. The cases of hyperfunctioning adenoma showed 83 per cent cured, 5 per cent markedly improved, 1 per cent slightly improved, 2 per cent not benefited, and 9 per cent dead from all causes.

It is fair to conclude from available statistics that the surgical treatment of hyperthyroidism cures about 75 per cent of all cases operated upon, that there is an operative mortality of at least 3.5 per cent, and a certain unknown percentage of patients that are inoperable.

*Roentgen-ray Treatment.* The roentgen ray has been used in the treatment of hyperthyroidism since 1898. It was only with the advent of the Coolidge tube, however, that it became possible to control the dosage to an extent that enables different workers to imitate or duplicate each other's results. Since 1915 the majority of roentgenologists in this country have used, with minor modifications, the dosage then recommended by Pfahler, and the results throughout the United States and Canada have for that reason been rather uniform. In 1915 Pfahler and Zulick[4] made a thorough review of the literature of the roentgen treatment of hyperthyroidism, and five years later Pfahler[5] presented an excellent summary of roentgen and radium therapy in this disease. He presents evidence based upon his own experience and that of careful workers in Europe and America which has led him to the conclusion that roentgen therapy offers an equal chance with surgery for the cure of hyperthyroidism, and it is his opinion that "radiotherapy is the best form of treatment for toxic goiter." Our own experience leads us to complete agreement with these conclusions. That surgeons in general not only do not accept these conclusions, but reject them almost entirely, is shown by the fact that Hertzler, in a recent book, "Diseases of the Thyroid Gland"[6] does not mention roentgen treatment of hyperthyroidism; that Crile, in his book, "The Thyroid Gland"[2] discusses the treatment only to condemn it; and that such statements as the following may be found in the writings of many prominent surgeons. "With *x*-ray treatments remissions may occur just as with other methods of treatment. Our experience has been failure, or but temporary benefit" (C. H. Mayo).

The reasons advanced by surgeons for rejecting the roentgen method of treatment are as follows:

1. It has not been proven that the roentgen ray produces permanent cure of the disease.

2. The time necessary for roentgen treatment increases the opportunity for cardiovascular and visceral changes to occur, and a certain number die of hyperthyroidism while taking the treatment.

3. Subsequent operation upon the gland is rendered more difficult because of adhesions between the muscles, capsule and gland.

4. The function of the thyroid may be so reduced that hypothyroidism will result.

The first reason given that roentgen therapy has not yet been proven of permanent curative value should be examined in the light of the fact that the roentgen-ray treatment of hyperthyroidism is still in its infancy. It has been only since the advent of the Coolidge tube that we have been able to control dosage, and only since 1915 that there has been any uniformity in the method of treatment employed. Surgeons have been operating upon patients with hyperthyroidism for more than forty years and have been able to amass a great volume of statistics which prove the value of surgical treatment. The roentgen method is now somewhat in the situation of surgery between 1900 and 1905, when the so-called medical treatment was that usually employed; the burden of proof at that time rested upon the surgeon. Roentgenologists are only now becoming able to point to permanently cured cases of hyperthyroidism, and cannot yet speak in terms of definite percentages, because sufficient time has not elapsed. In the very nature of the case it is still impossible to offer definite proof of the percentage of patients that are cured by roentgen treatment, but many cured cases are now being reported. There is no doubt that the results already obtained justify the extended use of this method, and it seems certain that the results will be greatly improved as roentgenologists gain experience in management of this disease and as more accurate dosage becomes possible by improvement in apparatus and technique.

That neither the surgical nor the roentgen-ray treatment of hyperthyroidism is perfect in its results may be inferred from the following quotations. The first is taken from Crile's "The Thyroid Gland"[2] and the second from an article by H. M. Jones.[7] Crile says: "It is significant to note that many cases that come to operation have had *x*-ray treatment." Jones says: "It is significant to note that many patients who come in for *x*-ray treatment have previously been operated upon."

The danger that the hyperthyroidism will increase and produce further damage while the roentgen treatment is being carried out is probably not great. In practically all the cases that are affected favorably by the roentgen rays the symptoms will begin to abate after about a month, that is, after the second treatment. Our experience indicates that five or six treatments is about the average number required. The first four are given three weeks apart and the next two at intervals of one month. The total time of treatment is thus less than four months. After the fourth treatment it is an almost invariable rule that the basal metabolic rate decreases steadily, the weight increases and the entire clinical picture is one of rapid improvement. In the rare case that does not follow this course, we have found it of little or no value to proceed with the treatment after the sixth application. These patients are kept under observation for about three months if the basal metabolic rate is not excessive and the general symptoms not severe. Great improvement may take place during this time and the condition may be such at the end of the period that a cure may be hoped for by further roentgen treatment. There remains, however, the occasional patient who shows little or no tendency to improve after the fourth treatment and it seems to us much the better course with such cases to advise thyroidectomy rather than to proceed further with the roentgen treatment.

If this course is followed, there will probably never be any basis in fact for the surgeon's third objection, that difficulty of operation is increased by roentgen therapy. There is abundance of testimony by surgeons that operation upon the thyroid gland is rendered less difficult by previous roentgen therapy because of the reduced vascularity. It is safe to say that such would always be the case if the roentgen treatment were kept within the bounds indicated above, and beyond which there is probably nothing to be gained.

The fourth objection to roentgen therapy—that it is likely to produce hypothyroidism—can be dismissed as of no practical importance. If this were a real danger, it would constitute a strong argument for the efficacy of the roentgen rays in reducing thyroid activity. If it ever was a danger of any moment, it can be avoided almost with certainty by controlling the treatment with the basal

metabolic rate. There is certainly no more danger of this accident after roentgen therapy than there is after thyroidectomy done by the average surgeon.

There would be no excuse for offering another method of treatment in this disease if surgery were free from danger, and if it always cured the disease.

Crile minimizes the danger of operation, stating that under the type of surgical management used by him the operative risk may be largely disregarded. The risk is real, however, and cannot be so lightly set aside in the face of the published statistics from many sources. We have quoted above statistics from the Mayo Clinic showing an operative mortality of 3.39 per cent in exophthalmic goiter, and we may fairly assume that the percentage is higher throughout the country. There is also a small but very certain mortality in the lesser operation of ligation of a thyroid vessel. The permanent cures effected by surgery may be judged by the figures quoted above from the Mayo Clinic, with 65.8 per cent of cures in exophthalmic goiter and 83 per cent in toxic adenoma.

The advantages of the roentgen method of treatment are (1) its freedom from danger, (2) its ease of application with a minimum of inconvenience and loss of time to the patient, (3) its availability in inoperable and postoperative cases.

We will conclude by stating briefly what we consider the essential points in the general management and treatment of a patient with hyperthyroidism:

A complete history is obtained and a thorough physical examination made. If a provisional diagnosis of hyperthyroidism is justified, the basal metabolic rate is determined. Inquiry is made with regard to living conditions, occupation, etc., and decision made as to whether the occupation may be continued or whether the patient should rest at home or in the hospital for a time. Careful instructions are given about the diet and amount of exercise to be taken. Sources of focal infection are sought and if the patient's general condition permits, they are removed.

Roentgen treatment is given over each lobe of the thyroid and over one area over the thymus region. We have not yet followed Pfahler's suggestion to cover the larynx, but have had two cases of troublesome hoarseness following treatment and think it would be well to use this precaution. The following are the essentials of our technique: 5 ma., at a 9-in. gap for six minutes over each area at 8-in. distance through 5 mm. of aluminum. Three successive doses are given at three-week intervals. The basal metabolic rate is again determined about three weeks after the third treatment. In a few cases no further treatment is necessary, but usually the rate is still somewhat elevated, and a fourth treatment is given at the usual three-week interval. Thereafter treatment is given at monthly intervals until two, or at the most, three more have been given. The necessity for these additional treatments is carefully considered in the light of the general condition of the patient and the basal metabolic rate.

We have treated 114 cases of hyperthyroidism and sufficient time has elapsed to state that 32 of these cases are cured, as indicated by their clinical condition and continued normal basal metabolic rate. Three patients have died: one of influenza, one of pulmonary tuberculosis, and one of hyperthyroidism. Four cases have been operated upon after roentgen treatment, but in only one of these was the roentgen treatment given sufficient trial. Six of our cases had had previous lobectomies or thyroidectomies. Two of these postoperative cases have returned to their work apparently well; one had permanent cardiovascular and visceral changes and is probably a permanent invalid, but her hyperthyroidism has subsided; the other three are recent ones and are doing well under treatment.

Twenty-four cases are greatly improved and will probably need no further treatment.

Three cases are clinically well, but after eighteen months or more they continue to have a somewhat elevated basal metabolic rate—from plus 22 to plus 28 per cent. We are urging further treatment in these cases, but have not yet convinced them that it is necessary.

In one case we have given a second series of three treatments because of moderate return of symptoms. This was followed by their prompt disappearance.

The results have, on the whole, been satisfactory, especially when we consider the fact that a number of these cases were of very severe type with basal metabolic rates above plus 100 per cent, some quite inoperable, and others operative failures.

## SUMMARY

1. Comparison of the results obtained in treatment of hyperthyroidism by surgery and the roentgen ray indicates that these two methods are probably about equal in the percentage of permanent cures.

2. Patients with hyperthyroidism should first receive roentgen treatment, and have thyroidectomy only if they fail to respond to this treatment.

3. The general management of patients with hyperthyroidism is of prime importance whether the ultimate treatment is to be the roentgen ray or surgery.

## BIBLIOGRAPHY

1. Pemberton, J. deJ. The surgical management of toxic goiters. *Boston M. & S. J.*, 1922, clxxxvi, 244–254.
2. Crile, Geo. W. The Thyroid Gland. Philadelphia, 1922.
3. Judd, E. S. Results of operations for adenoma with hyperthyroidism and for exophthalmic goiter. *Ann. Surg.*, 1920, lxxii, 145–151.
4. Pfahler, Geo. E., and Zulick, J. D. The treatment of exophthalmic goiter (Basedow's or Graves' disease). Am. J. Roentgenol., 1916, iii, 63.
5. Pfahler, Geo. E. Treatment of hyperthyroidism by radiation. *Med. Clin. N. A.*, Nov., 1921, v, 853.
6. Hertzler, Arthur E. Diseases of the Thyroid Gland. St. Louis, 1922.
7. Jones, H. M. Control of *x*-ray therapy in hyperthyroidism by basal metabolic test. *J. Radiol.*, Mch., 1922, iii, 85–92.
8. Boothby, Walter M. Adenoma of the thyroid with hyperthyroidism (thyrotoxic adenoma). History of the recognition of this disease as a clinical entity. A study of the symptomatology with basal metabolic rates. *Endocrinology*, 1921, v, 1–20.
9. Boothby, Walter M. The basal metabolic rate in hyperthyroidism. *J. Am. M. Assn.*, 1921, lxxvii, 252–255.
10. Deaver, John B. Hyperthyroidism from surgical point of view. *N. York M. J.*, Feb. 12, 1921, cxiii, 265.
11. Halsted, William S. The operative story of goiter. *Johns Hopkins Hosp. Rep.*, 1919, xix, Fasc. 2.
12. Kendall, Edward C. Some physiologic and chemical properties of thyroxin. Mayo Clinic, vol. xiii, Philadelphia, 1921.
13. Lichty, John A. Treatment of diseases of the thyroid with special reference to so-called hyperthyroidism. Am. J. Roentgenol., Dec., 1919, vi, 608.
14. Mayo, C. H. Adenoma with hyperthyroidism. *Ann. Surg.*, Aug., 1920, lxxii, 134.
15. Mayo, C. H. The thyroid and its diseases. *Surg., Gynec. & Obst.*, 1921, xxxii, 209–213.
16. McGuire, Stuart. Basal metabolism as an aid in diagnosis, prognosis, and treatment of hyperthyroidism. *J. South. Car. M. Assn.*, May, 1920, xvi, 107.
17. Means, J. H., and Aub, J. C. Basal metabolism in hyperthyroidism. *Arch. Int. Med.*, Oct., 1919, xxiv, 404.
18. Means, J. H. Hyperthyroidism—toxic goiter. *Med. Clin. N. Amer.*, Jan., 1920, iii, 1077.
19. Plummer, Henry S. The interrelationship of the function of the thyroid gland and of its active agent, thyroxin, in the tissues of the body. *J. Am. M. Assn.*, 1921, lxxvii, 243–247.
20. Sturgis, C. C., and Tompkins, E. H. Study of correlation of basal metabolism and pulse rate in patients with hyperthyroidism. *Arch. Int. Med.*, Oct., 1920, xxvi, 467.
21. Tyler, A. F. Roentgenotherapy of the thyroid. Am. J. Roentgenol., Jan., 1922, ix, 25.

## DISCUSSION

Dr. Pfahler. I can confirm everything Dr. Christie has said in his paper. I have treated about 200 cases. Three were operated on, and I am not willing to admit that they had to be operated on. In fact, I think they did not. One of these 3 cases was operated on because the patient was in the hospital and it meant too long a stay in the hospital to wait for cure by the *x*-rays. The other 2 cases were not willing to wait for results and were operated on. One patient developed recurrence which could not be controlled, and the patient died. Another patient, a very acute case, showed no response and died about a month after beginning treatment. That patient, however, was inoperable from the beginning.

I would urge the importance of covering up the arytenoid cartilage of the larynx. In that way you will prevent some of these disagreeable attacks of hoarseness which sometimes develop.

The technique which Dr. Whidman and I have been using is very similar to the one described by Dr. Christie. We usually treat through four areas: one, having the patient lie on back, dividing into median line, sending dose across from left side directed downward so as to catch the thymus, and likewise from the opposite side; then turning patient on face and giving two doses from behind, also directed downward. That enables us to reach the deeper portions of the gland. We usually begin with 12-min. exposure at 10-in. distance, 6

mm. filter, 5 ma., 9-in. spark-gap. After the first exposure we usually reduce the time to 10 min. We have not treated any with the high voltage, and I do not think we will. The results are so uniformly satisfactory to everyone that we have no reason to change, and the fact that the number of patients is continually increasing and that patients are constantly referring new ones, shows that the work is probably based on a solid foundation.

As I say, there have been only one recurrence and two deaths out of approximately 200 cases, and in the case of one of these deaths the patient could not be operated on. Therefore, we can bear out the statement made by Dr. Christie that there is no mortality from the treatment itself.

Dr. Dunham. I wish to add my appreciation of this magnificent paper of Dr. Christie.

There is only one point on which I would take issue, and that is the time of stopping treatment. He wants to stop after five or six treatments. Personally, I do not think that is enough. I know my failures would have been numerous had I followed that regime.

The point I wish to bring to your attention is that as *x*-ray men giving a new treatment we have been too much on the defensive. We have listened very keenly and properly for a long time to the criticisms of the surgeon, but *x*-ray treatment of hyperthyroidism has reached the point where we do not have to apologize; when we can stand and say that many more cases have to be treated by *x*-ray after operation than have to be operated on after *x*-ray treatment.

I have had 2 cases operated upon since 1903 by surgeons. They absolutely were taken away from me by the surgeon who told them that if they had another treatment, he would not think of operating.

I do not know the malignant thyroids. I cannot tell you how many patients might have had this, except for the treatment, or how soon I am going to have such a case in spite of the treatment, but I have had no such case.

What I have had is cases that have become temporarily insane—they went all to pieces. One had to be confined, but that case improved and got better, the tumor, tremor and exophthalmus almost disappearing. He is so much better that his physician advises against further *x*-ray as well as operation. He is not cured. Fortunately, none of my cases so far have died. There is a long series over a long time that have become practical cures. The cases which have followed my advice represent what I estimate as 100 per cent cures.

Two cases have gotten away from me because they were stolen—not because they had any right to go, not because I sent them to the surgeon, and not because they particularly wanted to go. It was because they were frightened into going, and that in itself is a crime. Cases of toxic goiter should never be frightened. Rest and everything else is a small matter compared to the avoidance of fright in a patient suffering from hyperthyroidism. Scare them, and the basal metabolic rate shoots up in an alarming manner.

Another thing I want to call to your attention. That is that you should not accept young girls for treatment who have just a little enlargement of the thyroid and rapid pulse. These symptoms become adjusted in a little while, and they do not need *x*-ray treatment or surgery. There are a number of such cases being operated on today by our surgeons, and I want to say to you that I believe the surgical attitude is something we ought to look into and resent.

The basal metabolic rate is one of the most valuable tests we have—that and the pulse. Plummer has called to our attention the cycles through which these cases pass—better; worse; better; worse. At one time you get a basal metabolic rate which is way down and you think the case does not need more treatment, and in a week or ten days it goes up again. So watch the phase of the cycle in which you examine your patient. That patient requires not only the management of which Dr. Christie speaks, but also the strength of your personality and assurance from the time that you take charge of the case until treatment is complete. Encourage your patient, and have him come back to you the very moment he is discouraged, even if it is not time for another treatment.

The treatment of exophthalmic goiter and hyperthyroidism by the *x*-ray is as marvelous as anything I know, next to the treatment of carbuncles, and I believe that no case should be operated upon.

Dr. Gray. I think this paper, if published in the Southern Medical Association Journal, will be the most valuable of any article ever published by that Journal; that is, so far as roentgenology is concerned.

I can see in this paper the processes that took place in the mind of Dr. Groover, who was chairman of the Radiological Section of the Southern Medical which met in Chattanooga last November. We had a joint meeting of the radiologists and the surgeons. Dr. George Holmes was scheduled to hold up the side of radiotherapy in the treatment of the thyroid, and Dr. Crile, the surgical side. Dr. Crile opened the discussion. After describing his procedure and stating the results, he threw on the screen a chart of 1,000 operations for

hyperthyroidism; 500 were thyroidectomies and 500 were ligations, with a mortality of 5/10 of 1 per cent about evenly distributed between the two.

He apologized for the next slide he showed and stated that he did not show it for the appearance of the skin but in illustration of the fact that *x*-ray made a thyroidectomy much more difficult. It happened, however, that the slide he showed was a typical exophthalmic goiter with bulging eyes, etc., but the skin below the neck, (well below the thyroid) extending to the middle of the chest had been terribly burned. The next slide was similar.

These were illustrations of the difficulty surgeons experienced in operating on thyroid glands after *x*-radiation, *but not for the purpose of showing any bad effects on the skin.*

The vast majority of the audience were surgeons. Dr. Holmes came on with his usual modest manner and gave the results of the investigations of the Thyroid Commission of the Massachusetts General Hospital.

Nobody was allowed to ask Dr. Crile how many of the cases which he had operated on had come to *x*-ray treatment afterwards. In my own experience I have had many more post-thyroidectomy cases to treat than I have had go from me to the surgeon.

I think I was one of the earlier men to treat hyperthyroidism; and in Dr. Pfahler's report there are several cases that I treated before we began filtration. I do not know how many cases I have cured permanently. I began the treatment back in the early days.

My trouble is that the patient is referred to me by a surgeon perhaps because some pressure has been brought to bear upon him for some other treatment than surgery. The patient is his, not mine, and I am serving under him in a way, and if improvement is not as he thinks it should be, he takes the patient and operates.

I have had some cases that I myself thought were very much better for operation than for radiation.

My technique is almost identical with that of Dr. Christie, except for a little difference in distance; I work at 10 inches and give a little more time.

I have in mind one of the best results I have ever obtained—a young woman of about thirty-eight years of age, who had had a lobectomy. She was temporarily relieved, but a recurrence took place and she was the most miserable individual I have ever seen; she had little or no exophthalmos but was very nervous; in fact, there was a question of sending her to an asylum. When she came to the office she could not endure having a light protection shield touch her face. About six treatments have gotten that young woman into the condition that enabled her to say to me just two weeks ago that she thought her life had been absolutely saved, as well as her mentality, by the treatment I had given her.

Dr. Christie (closing discussion). I would like to mention a point spoken of by Dr. Dunham. I over-emphasized in my paper that we tried to stop at about the fourth treatment. We do not always stop at four treatments, but that is one of the great advantages of having general observation of your patient. If you will watch your patient with great care and have a basal metabolic rate done often enough, and watch the pulse and weight, you can afford to wait a little while. It is just at this time, about the fourth treatment, that we are likely to be most discouraged in the treatment of this disease. If we wait, we will find the patient improved and then it is not necessary to give treatments so often. If we persist in giving numerous treatments, we will get the skin changes that come with the treatments, and if it happens that the case finally does go to operation, then we get fibrosis, etc. We must admit that, I think.

The most important point to be emphasized is that the general management of cases must come home to us, and we must take our cases in charge and actually treat them ourselves, or at least see that they get proper general management; that we do not permit the internist to send them for a dose of *x*-ray, and ignore them afterwards. We are apt to have a great many failures and bring *x*-ray into disrepute by such means.

# PROTECTIVE FACTORS IN MODERN HIGHLINE X-RAY WORK

BY ALBERT SOILAND, M.D.

LOS ANGELES, CALIFORNIA

THE incentive for American participation in high voltage *x*-ray work came from Europe where, during the late war, intensive studies were undertaken along this line. Among achievements of those who have contributed to this literature, Dessauer's work may be alluded to. He has built up ample transformers, and from these has measured wave-lengths, computed intensities, plotted absorption curves, and calibrated various filters. He has also described fully the biological reactions with varying intensities up to the capacity limit of his apparatus. The result of four years' clinical work abroad with similar transformers has been highly stimulating to many medical men and is, in a large measure, responsible for the active interest now being taken in broadcasting this work in America.

Following established custom, the American scientist was not satisfied with the voltage obtained by his European confreres, so he immediately bent his energies to furnish us with apparatus which materially increases the volt ratio over that used abroad; in fact, the voltage obtained is even higher than that which can be accommodated by the present types of hot cathode tubes.

The next factor, then, which engineering skill must face is to produce successfully tubes which will enable us to use the available high line. Voltages over 200,000, owing to the line stress, have the awkward habit of producing electrodynamic surges from occasional high peaks, acting very much like cross seas in a heavy swell, the usual result being a punctured tube.

The subject of high voltage *x*-radiation is already assuming such magnification that, in a single short contribution, only a few fundamental facts can be considered. The evolution of this new therapy will have to be followed slowly, carefully and with infinite patience. It will take years before one can be at all sure of the physiological responses, delayed reactions, and terminal effects. Our greatest efforts should be directed toward a standardization of procedure, so that a compendium of reliable data may in time be available.

One of the outstanding factors in radiology today is the problem of adequate protection, not only from the energy of radiation, but also from the high potential electrical current. This latter takes on added importance when it is applied to the installation of apparatus that requires a voltage of 200,000 or more, for its operation. To protect securely both patient and operator from any danger of contact with this line is a problem of no small proportions. Especially is this matter pertinent owing to the multiplication of high voltage installations now going on in all parts of the world. It is true that death has not been frequent from contact with one side of the line carrying energy to the universal type of tubes, running all voltages not in excess of 100,000, although you will recall that there have been deaths from such contact. It is when this voltage is more than doubled that the question assumes a sinister aspect, and this is the situation which you and I will be called upon to meet and conquer. One also must fully realize his personal obligation to humanity when creating, by means of higher voltages, an energy as yet unmeasured, both qualitatively and quantitatively; an energy termed by the writer "super-radiation," our knowledge of which is at present pitifully elementary. We do know, however, that a single vacuum tube, under such stimulus, is capable of projecting an enormous volume of *x*-radiation very closely related in ethereal form to the gamma radiation from radium.

Today, modern engineering skill has produced for us apparatus which with mechanical certainty will generate radiation energy practically unlimited in range and scope. The apparatus is so constructed and assembled that, by merely turning a switch, this unseen and insensible energy may be dispersed through space, there either to renew or destroy life, according

to the operator's ability to control the forces at his command. Knowing this, it is paradoxical that there is in this country no specific law to prevent any person, irresponsible or otherwise, who has sufficient funds with which to purchase such an apparatus, from using the same indiscriminately. It is, therefore, imperative that we who are vitally interested in this work should protect it from all hazards to the best of our scientific and personal ability.

The transformer which energizes an *x*-ray tube for deep treatment has a potential force which varies from two to three hundred kilovolts. To come into contact with any part of this high line would mean, almost invariably, sudden death. This takes on a more somber meaning when applied to many of the present-day high tension installations which are so mounted that a patient under treatment can, by a careless movement, come into actual contact with the line at several points.

The employment of high voltage *x*-rays in routine work brings with it a twofold responsibility. In the first place, both the patient and the operator must be shielded from the effects of the more powerful *x*-rays we are now employing, and secondly, all possible opportunity must be excluded for either patient or operator coming into contact with the high voltage line.

Preventing the wandering of stray rays and confining the radiation to the desired path is relatively simple; but it requires lead, in fact a great deal of lead. To obtain adequate metallic protection equivalent to ¼ in. lead, and to allow space for operating up to 300,000 volts requires about 1,000 lbs. per tube.

Protecting the patient or operator from the high tension line can only be accomplished with perfect safety in one way. The high tension wires must clear all objects by an air space equivalent to ½ more than the full sparking voltage, and a grounded conducting shield must intervene between the wiring and all persons who may approach it.

This is exceptionally true of the writer's installation where auto-transformer control is used on the high voltage apparatus, and no resistance whatever is included in the circuit. This arrangement has made it possible to maintain effective standardized operation, and several tubes may be run at once from the same apparatus without appreciable line fluctuation, but it makes necessary an even greater attention to safety, as the power of an electrical shock from this equipment is almost beyond calculation.

To meet the condition of full *x*-ray protection from any high voltage operation, so much lead is required that it appears to the writer impractical to attempt a mobile tube unit; therefore, the patient is moved and the tube mounted stationary.

A happy combination of high voltage and *x*-ray protection has been devised by placing the tube under a bridge deck and liberally surrounding it with lead. The rays are emitted upward through a variable orifice and, through suitable filters, reach the patient. There is also a diaphragm opening provided in the front face of the deck, through which, by simply rotating the tube, the stream may be directed to a patient who is in the sitting posture. It is advisable, however, to make use of this portal only when absolutely necessary in order to avoid pointing the ray beams toward the operator or observer. It is, of course, possible to protect against this contingency also, but it adds to the existing complications.

It will be found that nearly all conditions can be met successfully by placing a movable couch, which supports the patient, above the tube. This gives a constant skin target distance. A small plumb bob suspended from a swinging bracket above the patient serves to locate the focal point of the tube, and the couch can be readily moved until the area desired is in juxtaposition with the central ray.

Not the least important in this whole arrangement is the total freedom of movement permitted both patient and operator, there being no possible chance of a contact with any part of the high line. In addition, the tube is suspended in a spacious chamber connected with outside air and completely insulated from operating room. This obviates the necessity of fans, and

prevents any of the noxious oxides or other electrical adjuvants from reaching the patient; a point to be remembered when thinking of the usual position of the patient lying under an ordinary overhead installation, particularly of the heavy and complicated varieties.

In the writer's laboratory, tubes are working at a potential pressure of 220,000, measured and checked by the engineers' standard sphere gap device for effective voltage, in addition to the available penetrometers, iontoquantimeters and similar instruments. It is possibly needless to say that the most painstaking care is exercised when employing this voltage under working conditions. In addition to a personal attendant at the board, we employ a reliable time-switch arrangement connected with two loud-toned gongs which automatically strike when the treatment period is concluded. The reason for having two such gongs is merely in case one should mechanically fail to operate.

Another important factor we employ is a reliable magnetic circuit-breaker. This may be adjusted so as to break contact the moment any unexpected surge or high peak wave develops. This circuit-breaker may not only save one from physical injury, but also obviates the necessity of replacing costly tubes and broken-down transformers.

Much profitable time could be spent on an elaboration of many of these factors, but as you are all familiar with most of the protective and safety devices which enter into the installation and operation of a modern plant for deep roentgen therapy, it will be omitted.

It is much too early to state positively what we may expect in deep malignancies, and sufficient time has not yet elapsed to enable us to obtain enough cases for final determination. Conservatively, it may be stated that from present indications we have more than mere theoretical grounds for optimism. If, in the course of this preliminary presentation, you have realized that the subject under discussion merits not only your respect, but also your unqualified interest and support, then a scientific future for deep roentgen therapy may be confidently expected.

# THE BACTERIOLOGY OF IRRADIATED TONSILS*

BY H. J. ULLMANN, M.D., AND F. R. NUZUM, M.D.

Santa Barbara Cottage Hospital,

SANTA BARBARA, CALIFORNIA

IN 1920 Murphy[1] reported tonsils free of hemolytic cocci three weeks after exposure to the roentgen ray. In all cases either hemolytic streptococci or staphylococci had been originally found. The cultures were made from the crypts and grown on blood-agar. Later, in 1921, he and Witherbee[2,3] reported that the common organisms found in the throat were unaffected by roentgen irradiation, but 30 of 36 cases showing hemolytic streptococci and staphylococci became free from these organisms by the fourth week after treatment. Price[4] states that the roentgen ray disinfects tonsillar tissue. Hickey[5] reports fifteen cures in 19 cases of diphtheria carriers.

In bacteriologic studies of infected tonsils, streptococci have assumed more prominence than other organisms. Further work which has divided streptococci into hemolytic and non-hemolytic varieties has demonstrated that the hemolytic organisms are the virulent ones and the ones associated with clinical conditions.

The most complete classification for streptococci is that of Holman,[6] which divides these organisms into sixteen groups, eight of which are hemolytic and eight non-hemolytic. We have, however, followed a less elaborate but very practical classification, that of T. Smith and J. H. Brown,[7] which divides all streptococci into four groups, depending upon their manner of growth in blood-agar plates. Alpha hemolytic streptococci are characterized by a small zone of brown or green discoloration immediately about the colony.

* Read at the Twenty-third Annual Meeting of THE AMERICAN ROENTGEN RAY SOCIETY, Los Angeles, Calif., Sept. 12–16, 1922.

Alpha prime hemolytic streptococci have immediately about the colony a small area of incompletely hemolyzed red blood-cells. Beyond this is a ring of complete hemolysis. Beta hemolytic streptococci is that group which has a zone of complete hemolysis immediately about the colony. This clear zone varies from 1 to 4 mm. in width. The fourth, or final division of streptococci, is the non-hemolytic colonies. They appear as small, dark dots, and produce no change in the adjacent corpuscles of the media.

*Technique.* Our procedure in cultivating the tonsils was as follows: A sterile swab was drawn across the tonsillar surface and then placed in 1 c.c. of sterile physiologic salt solution. A platinum loop bent at a right angle near its distal end was introduced into a crypt, withdrawn, and dipped into a second tube containing 1 c.c. of sterile physiologic salt solution. A few drops of each of these inoculated solutions were poured into Petri dishes and 12 c.c. of liquid blood-agar added. The Petri dishes were gently agitated until the salt solution had become well mixed with the blood-agar and then put aside to cool, after which they were incubated for forty-eight hours. Streaked blood-agar plates were made from the same material and incubated. The plates were read at the end of twenty-four and forty-eight hours. Bile solubility and the various sugar media were employed in making a final classification of doubtful colonies.

*Results.* In a series of 218 pairs of pathologic tonsils that had been removed and examined by the above method, one of us (Nuzum)[8] found hemolytic organisms in 96.1 per cent. Of the hemolytic organisms, beta streptococci were present in 86.1 per cent; alpha in 25 per cent; and alpha prime, or streptococci viridans, in 32 per cent. This is in agreement with recent findings of Davis[9] and of Pilot and Pearlman.[10]

Because of the clinical importance of these organisms and the frequently reported clinical benefits resulting from irradiation of tonsils, the authors have attempted to determine whether the results of radiation of tonsillar flora were not more or less selective and affecting principally the pathogens, and if so, in what percentage could bacteriologic results be expected. The number of cases studied is too small to draw any hard and fast conclusions, and therefore this paper is intended to be in the nature of a preliminary report.

Forty patients said clinically to have infected tonsils have been studied. Prior to irradiation, beta hemolytic streptococci were isolated in each instance. Seventy-five per cent had alpha organisms, and 80 per cent had alpha prime organisms. Non-hemolytic or gamma streptococci were found in 40 per cent of the instances. The explanation for all types being found in nearly every case is that many cultures were made from time to time, and during the course of cultures the various organisms were found.

Of the 40 patients studied in this preliminary manner, all of whom received treatment, 20, or 50 per cent, for various reasons did not receive enough treatment to warrant making a final decision as to the effect of the roentgen ray upon the bacterial flora. Twenty did receive what was considered sufficient treatment. In 20 per cent of this number, marked gross change occurred in the tonsils. In each instance this consisted in the marked shrinking of the tonsil; the crypts became everted and shallow; the mucous membrane covering the tonsil pale gray and smooth; and the cultures from these tonsils became negative for all types of streptococci. In addition to the tonsils which underwent marked gross change, a further group of 35 per cent became negative for beta hemolytic streptococci. These tonsils remained free from these organisms up to six months following the last treatment. None have been cultured for a longer time than that. In several instances the tonsils did not become immediately free from beta organisms, even for so long as one month after the last treatment, but when the patients were cultured at the end of six months they were found to be free. The remaining 45 per cent of the patients in this group presented no marked gross change in the tonsils, and no change occurred in the bacterial flora. Beta hemolytic organisms were present in every

instance and alpha and alpha prime organisms were frequently found.

The technique, with the exception of 6 cases, was that recommended by Witherbee. With these 6 the technique was as follows: K.V.P., 200; F.S.D., 50 cm.; port 9 cm. sq.; filter, Cu 1 mm., Al 1 mm.; ma. min., first sitting, 25; second sitting, 35; third and fourth sittings, 50. Forty-eight hours elapsed between the sittings. One patient received two series with an interval of three months, the first with the Witherbee technique, the second with the one described above. In these 6 cases the results were exactly 50 per cent successful. One of the unsuccessful cases was that of the patient who had had the two series.

No relation between the clinical and bacteriological results was observed. Some, whose throats had become free of streptococci, said that they could see no difference in the condition of their throats following treatment. Others, who still harbored pathogens, were highly pleased with the results. The most interesting of these was the patient who had received two series. She states that a tonsillitis had always followed or accompanied any acute infection of the upper air passages, but that following radiation she has had several more or less severe "colds" without tonsillar involvement. This in spite of our failure to obtain results from a bacteriological standpoint. It would seem that some other explanation than that of sterilization must be found for the clinical benefits observed in the treatment of infected tonsils by the roentgen ray.

## SUMMARY

Of 20 cases receiving roentgen irradiation of the tonsils, 20 per cent underwent a marked gross change, became much smaller, and remained free from beta hemolytic streptococci. An additional 35 per cent became free from beta hemolytic streptococci and remained so for six months after treatment. Forty-five per cent still harbored these organisms following the entire series of exposures. No difference in results was seen between those treated by the Witherbee technique and those treated by the technique described, and no relation between clinical and bacteriological results was observed. The series reported is too small to draw conclusions from, and it is hoped that a larger series will be available in the near future.

## BIBLIOGRAPHY

1. Murphy, James B. Effect of small doses of roentgen-ray on lymphoid deposits. *J. Am. M. Assn.*, June 19, 1920, lxxiv, 1738.
2. Murphy, James B., Witherbee, W. D., Craig, S. L., Hussey, R. G., and Sturm, Ernest. *J. Am. M. Assn.*, Jan. 22, 1921, lxxvi, 228.
3. Witherbee, W. D. The principles involved in the *x*-ray treatment of tonsils. *N. York Med. J.*, March 16, 1921, cxiii, 484.
4. Price, Byron Sprague. Treatment of adenoids and tonsils by *x*-ray vs. surgery. *Am. J. Electrotherap. & Radiol.*, Feb., 1921, xxxix, 81.
5. Hickey, P. M. Treatment of diphtheria carriers by means of roentgen ray. Am. J. Roentgenol., May, 1922, ix, 319–322.
6. Holman, W. L. Classification of streptococci. *J. Med. Research*, 1916, xxviii, p. 337.
7. Smith, T., and Brown, J. H. A study of streptococci isolated from certain presumably milk-born epidemics of tonsillitis occurring in Massachusetts in 1913 and 1914. *J. Med. Research*, 1915, xxxi, 445.
8. Hambrecht, L., and Nuzum, F. R. A correlated study of the indications for tonsillectomy and of the pathology and bacteriology of excised tonsils. *Arch. Int. Med.*, 1922, xxix, 635–642.
9. Davis, D. J. Hemolytic streptococci. *J. Am. M. Assn.*, Feb. 1, 1919, lxxii, 319.
10. Pilot, I., and Pearlman, S. J. Bacteriologic studies of the upper respiratory passages. *J. Infect. Dis.*, 1921, xxix, 47.

## DISCUSSION

Dr. Remer. I was very glad to hear Dr. Ullman say that often within a month you still find microorganism, and that it is frequently a month or two before those microorganisms have disappeared.

Dr. Ullman (closing discussion). The point that we are trying to bring out evidently is that while there is a difference in the susceptibility of the various organisms to radiation, the results obtained are more apparent than real. We have found in culturing that perhaps they will be sterile or nearly sterile for pathogens in early cultures, then clear again, then become sterile again. We have had some become sterile immediately, then become infected again. Before this can be answered I think we should take cultures of most of them that have been radiated every week or two for six months and see if there is not a normal curve. There is less bacteria at one time than at another. If the pathogens are more susceptible, then the patients who have received marked benefit should have fewer of those pathogens. There is no relation between the clinical results; some who are benefited still have pathogens, and vice versa.

# MEASUREMENT OF DOSAGE BY MEANS OF IONIZATION CHAMBERS*

BY WILLIAM DUANE, PH.D., SC.D.

Professor of Biophysics, Harvard University

BOSTON, MASSACHUSETTS

THE fact that different *x*-ray plants produce *x*-rays of different intensities and effective wave-lengths even though they may be running at the same voltage, as estimated by a sphere-gap, and with the same current through the tube, indicates that we must use a method of measuring something connected with the *x*-ray beam itself, if we wish to get reliable estimates of dosage.

Variations of 40 per cent and more in the intensity of the *x*-rays projected through the same filter and at the same distance from the tube with different machines are not uncommon, and in extreme cases one machine may produce twice as much *x*-radiation as another.

Without doubt, ionization chambers provide us with the most reliable methods of measurement, at present. Ionization chambers, however, are by no means perfect, and great care must be exercised in employing them.

I purpose, in this discussion, to describe the method we are using and to call attention to certain pitfalls into which one is apt to stumble, hoping that this may prove of use to those of you who are using ionization chambers, or who may be planning to purchase them.

When a beam of *x*-rays passes through a gas, such as air, it splits up the molecules of a gas into particles (called ions), some of which carry a positive charge of electricity, and others a negative charge. If left alone, these particles will recombine with each other, owing to the attraction of those charged positively for those charged negatively. By applying an electric force to the ionized gas, however, we can separate the positively charged particles from the negatively charged ones. For instance, suppose that the ionized air lies between two parallel metal plates, one of which is connected to the positive pole of a battery and the other to the negative pole. Under these conditions the positively charged plate attracts the negatively charged ions and draws them to it out of the air; similarly, the negatively charged plate attracts the positively charged ions and pulls them out of the air in the opposite direction. Thus, the battery produces a current of positive electricity through the air in one direction and a current of negative electricity through the air in the opposite direction. This current may be measured by a galvanometer suitably placed in the electrical circuit. We may use the electrical current as measured by the galvanometer as an indication of the intensity of the *x*-ray beam, for, in general, a strong beam of *x*-rays produces a larger number of pairs of ions than does a weak one.

The electrical current, as measured by the galvanometer, however, will not give reliable estimates of the intensities of *x*-ray beams unless in each case the electrical force acting on the ions suffices to remove practically all of them before they have time to recombine with each other. For example, if two equal and similar beams of *x*-rays pass through the air between the plates, the current will not be twice as great as that due to one beam alone, if an appreciable number of ions recombine with each other before they reach the plates; for with the two beams passing through the air, twice as many pairs of ions are produced per second as with one, and there is a much greater chance for the ions to recombine with each other. Those ions which recombine cease to produce their share of the electrical current. If, however, the electromotive force of the battery produces an electrical force on the ions of sufficient magnitude to remove practically all of them before they have time to recombine, then the electrical current due to two equal, similar beams of *x*-rays will be

* Read at the Midwinter Meeting of the Eastern Section of THE AMERICAN ROENTGEN RAY SOCIETY, Atlantic City, N. J., Jan. 25–27, 1923.

Discussion on this article will appear in June.

twice as great as that due to one of them alone. In this case we may take the currents as proportional to the intensities of *x*-ray beams.

If the electric force acting on the ions suffices to remove practically all of them before they can recombine, the ionization current is said to be saturated. Only saturated ionization currents should be used to measure *x*-ray beams.

In some ionization chambers great difficulty may be encountered in producing the saturated current; in other cases, a small battery may produce such saturation. I will illustrate these points by describing several experiments, in some of which I could easily produce a saturation current and in others of which the batteries at my disposal were not sufficient to saturate the current.

The diagram in A (Fig. 1) represents a hollow, metal cylinder, closed at one end, with a rod lying along the axis of the cylinder, the cylinder being joined to a battery and the rod to a galvanometer, and the other pole of the battery being connected to the galvanometer so as to form a complete electrical circuit. The cylinder and rod lie in a closed, glass vessel, as indicated.

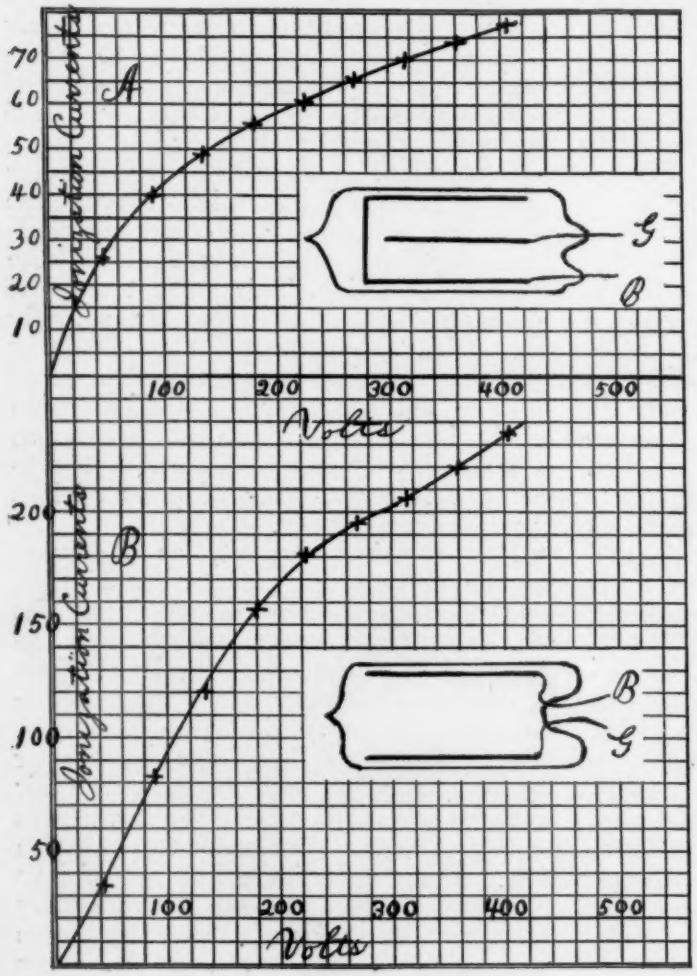



FIG. 1.

On sending a beam of *x*-rays through this ionization chamber and on changing the number of cells in the battery, that is, on changing the voltage applied to the cylinder, a number of different currents through the chamber were obtained. The curve in A (Fig. 1) gives the readings of the galvanometer at different voltages applied to the cylinder.

It appears that on increasing the number of cells in the battery, that is, on increasing the voltage applied to the cylinder, the ionization current, as measured by the galvanometer, keeps on increasing and does not reach a constant value. This means that, as the electrical force acting on the ions increases, more and more of the positive ions become separated from the negative ions before they have time to recombine. In this case, however, the electrical force applied was never sufficient to remove practically all of the ions before they could recombine with each other. In other words, a saturation current was not produced. An ionization chamber with these characteristics should not be used to measure *x*-ray beams. Cylindrical ionization chambers of this type should never be used, if the wire along the axis of the cylinder is very fine.

B (Fig. 1) represents an ionization chamber in which both electrodes are metal plates, one of them joined to the battery and the other to the galvanometer, as

above. In this case, on increasing the voltage applied to the plates the curve in B was obtained. This curve also indicates lack of saturation; for the ionization current continues to increase as the size of the battery increases. In this chamber the metal plates did not cover the entire sides of the glass vessel containing them and left corners in the vessel from which the electric force was unable to withdraw all of the ions. The design of this chamber illustrates again a kind of ionization chamber that should not be used.

the enclosed air. In this chamber the two outside plates go to the battery and the inside one to the galvanometer. The curve D (Fig. 2) indicates practically complete saturation by voltages above 20 volts. For saturation purposes this chamber represents the best type that I have examined. It is the one we are now using in our measurements of the intensities and effective wave-lengths of *x*-ray beams.

As indicated above, the question of saturating the ionization current becomes one of prime importance. No chamber

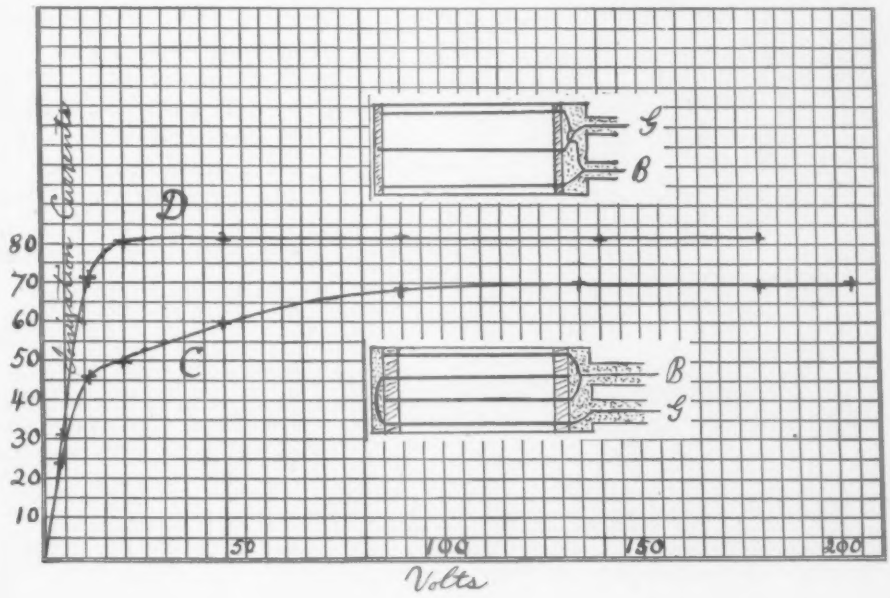



FIG. 2.

C in Figure 2 represents a chamber in which the parallel metal plates extended to the ends of the closed space, enclosing the air. They did not, however, extend to the sides of this enclosed space in a direction perpendicular to the diagram. Further, one of the plates joined, as indicated, to the galvanometer lay close to the metal sheet wrapped around the chamber for protection. The curve giving the current produced by different voltages indicates a much closer approach to saturation in this case than in the two preceding cases. In other words, the design of the chamber is better than those of the first two.

It is not quite as good, however, as that represented by D (Fig. 2) in which all three plates extend completely across

should be used or purchased unless it has been proved experimentally that the electric force produces saturation.

If saturation currents are not obtained, the readings of the galvanometer do not give reliable measurements of *x*-ray beams. Intense beams will produce relatively smaller deflections than they should, and, vice versa, weak beams of *x*-rays will produce relatively too large deflections. Perhaps some of the exceptionally high depth dose readings obtained by certain observers may be due to the fact that they were not using completely saturated ionization currents.

In order that we may compare with each other measurements made in different laboratories and in different clinics it is

desirable to have some standard unit of *x*-radiation and some standard method of measurement. The unit that I have been using for the last nine or ten years may be defined as: "That *x*-ray beam which would produce one absolute electrostatic unit of current in each cubic centimeter of air through which it passes, provided that the current has its saturation value."

Ionization chamber D (Fig. 2) does not appear to be suitable for measuring currents in terms of this unit, for two reasons. Firstly, a large part of the ionization arises from the secondary rays produced by the primary beam of *x*-rays in the plates and walls of the chamber. The secondary rays increase the ionization current and the readings of the galvanometer become, therefore, too high. This secondary ray effect may be greatly reduced by using some substance of low atomic weight in place of the metal plates. In the measurements of effective wave-lengths recorded in an article by Dr. Hunt and myself[1] we used sheets of thin paper with faint pencil-marks drawn across them. The plates, therefore, were very thin layers of carbon. A much better method, however, is that which I have previously employed to standardize the ionization chamber that we actually used. In this method I compared the current produced in the chamber to be standardized with that produced by the same beam of *x*-rays in a chamber so designed that the *x*-ray beam did not strike anything inside the chamber. Figure 3 represents one of these standard ionization chambers.

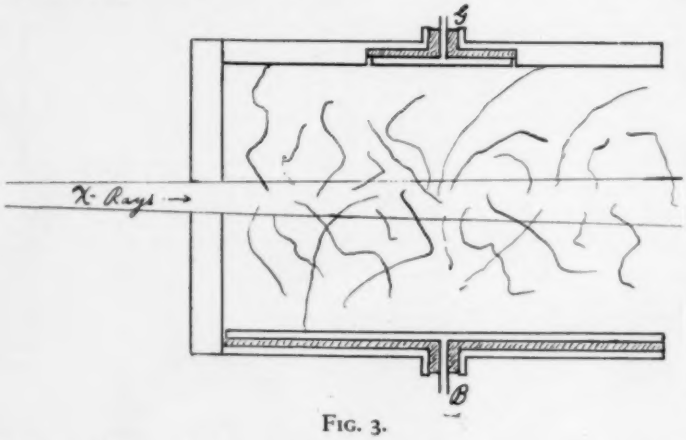



FIG. 3.

The *x*-ray beam enters the chamber through a hole of known area and passes between the plates without striking them. The plates are arranged in a manner similar to those in an instrument designed by Lord Kelvin and called a guard-ring condenser. One plate, connected with the battery through B, extends almost the whole length of the chamber. The other side of the chamber is divided into three sections. The two end sections are joined to the outside metal casing of the chamber, which, in turn, is connected to earth for purposes of protection. The middle section of the side, insulated from the casing, is connected through G to the galvanometer. The middle section has a certain definite breadth and draws its ionization current from an equal length of the *x*-ray beam. Since the *x*-ray beam has a definite, known cross-section, this means that the current going to the galvanometer comes from a certain volume of air. The volume of air in my instruments amounts to 25 c.c. If I get from this ionization chamber a current through the galvanometer of 25 electrostatic units, then each of the 25 c.c. of air produces one electrostatic unit and my beam has unit intensity, according to the above definition of unit beam. This is the standard ionization chamber that we use in calibrating the smaller chambers actually employed to measure the *x*-radiation during treatments.

There is one important point to be borne in mind in designing a standard ionization chamber of this kind. When *x*-rays produce ionization in a gas, a certain amount of secondary radiation is generated in the atoms and molecules of the gas by the primary beam. This secondary radiation produces a large amount of the ionization. The secondary rays, however, travel some distance from the path of the primary beam. They are of two kinds—corpuscular-rays and *x*-rays. The secondary *x*-rays are very penetrating and travel a long distance from the beam. The corpuscular rays, however, follow very crooked paths, somewhat as indicated in Figure 3, and only a few of

[1] *Phys. Rev.*, Aug. 1915, p. 166.

them penetrate to a great distance from the primary beam.

In 1905 I pointed out that a correction must be made for the rays that strike the walls of a vessel and for secondary rays coming from those walls, if one wishes to measure quantities of radium emanation by the ionization method.[1] The correction, as determined by my experiments, proved to be proportional to the ratio of the surface of the ionization chamber to its volume. As this ratio becomes very large for small volumes it indicates a certain disadvantage in using very small ionization chambers. Theoretically, a standard ionization chamber should be indefinitely large so as to include all the ionization produced by all the secondary rays. Practically, however, the vast majority of secondary rays travel along such crooked paths that they do not get very far from the primary beam. It is not, therefore, necessary for therapeutic purposes, at least, to use very large standard ionization chambers. Those that I was using nine years ago had volumes varying from 500 to 2,000 c.c., and this appeared to be sufficient for the *x*-rays that were being produced at that time in practice.[2]

In order to test the suitability of a standard ionization chamber it is necessary, firstly, to make sure that the ionization current is saturated, and secondly, that it includes practically all of the secondary radiation coming from the molecules of gas struck by the primary beam.

The curves in Figure 4 represent the ionization currents passing through two of our standard ionization chambers of different sizes when different voltages were applied to them. The curves indicate that the current in the smaller one became saturated at about 300 volts and that the current in the larger one became saturated at about 500 volts. When using either of these chambers I employ about 800 volts, so as to be sure of their saturation.

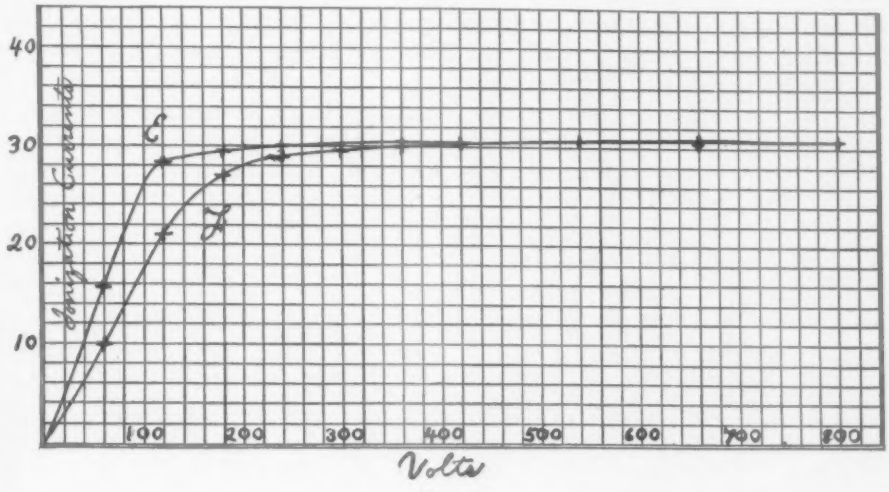



FIG. 4.

In the experiments represented by the curves in Figure 4 the voltage applied to the *x*-ray tube amounted to about 100,000 volts, and I was unable to detect any difference between the ionization current in the larger chamber and that in the smaller chamber. Either chamber, therefore, appeared to include practically all of the *x*-ray effect due to the secondary rays coming from the air in the chamber.

On using 200,000 volts, however, *x*-rays of much shorter wave-length were produced and the secondary radiation became more penetrating. In this case I found a difference of about 5 per cent between the currents through the two chambers. The larger chamber produced the larger current. On increasing the distance between the plates of the larger chamber, however, no perceptible increase in the ionization current occurred. It

[1] *Compt. rend. Acad. d. sc.*, and *J. de Physique*, 1905.
[2] Friedrich employs standard ionization chambers somewhat similar to those described here.

appears, therefore, to be large enough to use as a standard, even with rays produced by 200,000 volts. A distance of 10 cm. between the plates of an ionization chamber seems to be sufficient, if 200,000 volts are applied to the tube.

It is, perhaps superfluous to call attention to the fact that in estimating dosage (erythema dose, for instance) it is necessary to measure the effective wave-length of the beam as well as its intensity, for the amount of *x*-ray energy absorbed by the tissues depends upon the wave-length.

These wave-length measurements may be made with either a standard ionization chamber or with one of the smaller metal chambers. It appears, however, necessary to standardize the smaller chamber for wave-length measurements by comparison with the standard ionization chamber.

One important feature of the method I am describing lies in the fact that the reading of the galvanometer gives the intensity of the *x*-ray beam and not the total dose received by the patient. In order to get the total dose we have to multiply the intensity by the time of exposure.

The large, standard ionization chambers are not suitable for measurements of the intensity during a treatment. We invariably use one of the smaller ionization chambers to measure the intensity of the beam received by the patient several times during the treatment. We measure the intensity of the rays at the surface where they enter the patient's body, and also where they emerge. This gives us an estimate of the secondary radiation coming from the patient's body. The estimate, however, is too low. Estimates may be made by means of water phantoms, placing the small ionization chamber in the water itself. This estimate is always too high. The real dose received by the patient's skin lies between the two. We have obtained quite variable estimates of the secondary radiation from different patients made by measurements taken during the treatments themselves. The secondary radiation appears to depend not only upon the size of the portal of entry but also upon the size of the patient and upon the shape, content, etc., of the portion of the body radiated. In estimating erythema doses all of these factors must be taken into consideration. The safest method appears to be to make the measurements while the patient is actually being treated.

In many of the ionization methods of measuring dosage one determines the ionization current by timing with a stopwatch the passage of the leaf of an electroscope across a scale. In methods of this kind some difficulty often arises in determining whether the current is saturated or not. Very particular attention should be paid to this point.

I might mention, also, another very common source of error in electroscope measurements. In such measurements one should always determine the "leak" in the instrument. This should be done with the *x*-ray tube running: for the *x*-rays may produce ionization currents in the electroscope (if it is not completely protected), or in parts of the apparatus other than the ionization chamber itself. Corrections must be made for the "leak," when it exists.

If one makes percentage depth dose measurements in a water phantom, one should determine the "leak" with the ionization chamber at the surface of the water, and also, at the various depths used below it. Perhaps the best way to measure the "leak" is to place a thick sheet of lead over the opening that determines the cross-section of the *x*-ray beam (the lead being thick enough to stop practically all of the *x*-rays), and then to measure the current. The leak current must be subtracted from the ionization current (obtained after removing the lead sheet), the two currents being inversely proportional to the lengths of time during which the leaf in the electroscope moves from one point on its scale to another.

If one measures percentage depth dose, the following well-known formula may be employed to correct for the leak. With the ionization chamber at the surface of the water phantom let $L_0$ be the number of seconds corresponding to the "leak" (i.e., when the lead plate stops the *x*-rays) and let $T_0$ be the number of seconds corresponding to the ionization current (after the lead plate has been removed). Further let $L_1$ and $T_1$ be the

corresponding numbers of seconds, when the ionization chamber lies at any distance (say 10 cm.) below the surface. The formula for the percentage depth dose may then be written:

$$\text{P.D.D.} = \frac{\frac{1}{T_1} - \frac{1}{L_1}}{\frac{1}{T_0} - \frac{1}{L_0}}$$

If no correction be made for leak currents, and the depth dose is determined simply by dividing one length of time by another, a large error may be introduced. In one experiment that came under my observation the uncorrected depth dose amounted to considerably over 40 per cent with the tube running at approximately 200,000 volts. When the correction was made, however, the measured depth dose fell to about 34 per cent.

A great many important investigations have been carried on in connection with the question as to whether the biological effects of *x*-rays are proportional to ionization currents, when rays of different wave-lengths are used. One speaks of the biological dose. In particular cases biological doses are definite quantities. Before we can speak of a biological dose in general, however, it will be necessary to show by experiments that a large number of different biological effects are proportional to each other, when *x*-rays of different wave-lengths produce the effects.

# ISODOSE CHARTS

BY OTTO GLASSER, PH.D.

HOWARD A. KELLY HOSPITAL

BALTIMORE, MARYLAND

CHARTS and tables for the distribution of intensity at various depths in the human body have been worked out for the deep therapy treatment with roentgen rays for certain types of malignant and benign tumors of frequent occurrence. Friedrich[1] of Freiburg was one of the first to determine accurately the distribution of depth intensities; the most comprehensive work in this line was done by Dessauer and Vierheller[2] with their well-known intensity charts. Duane,[3] Bachem[4] and others have made valuable contributions to this work.

Many roentgenologists, working with the charts of Dessauer, have experienced certain difficulties in their use, especially when working with American transformers. These difficulties seem mainly to be present in measuring and comparing the coefficient of weakening $\mu_{water}$ with which the quality of the used *x*-ray bundle in the Dessauer charts is characterized.

For the most common technique of deep therapy treatment, the distribution conditions were, therefore, remeasured for American transformers (3 different types used) with both an accurate small horn ionization chamber and a photometric method in a large water phantom. The conditions were as follows: 200 kv. (peak), 0.75 mm. Cu + 1 mm. Al. filter, ports of entry 20 × 20 cm. (Fig. 1) and 10 × 10 cm. (Fig. 2), 50 cm. focus-skin distance. The effective wave-length according to Duane under these conditions was $\lambda_{eff.} = 0{,}15$ Å*u*, the coefficient of weakening according to Dessauer was $\mu_{water} = 0{,}180$; both were measured with the Dessauer-Bachem electroscope. The peak voltage at the tube was exactly recorded by means of a spectrogram, taken with the Seemann spectrograph. The results of the measurements are shown in Figure 1 and Figure 2.

In these diagrams, points of equal intensities are connected with each other. All intensities in the depth are expressed in percentages of the total surface intensity 100. The curves are called "isodoses," a name I gave first to these curves in connection with radium five years ago.[5] (The name is justifiable when a practical homogeneous *x*-ray bundle and a definite time of radiation are adopted.)

These isodose charts express all the known (Friedrich, Dessauer etc.,) features of intensity distribution in the depth: main intensity (usually) in the central

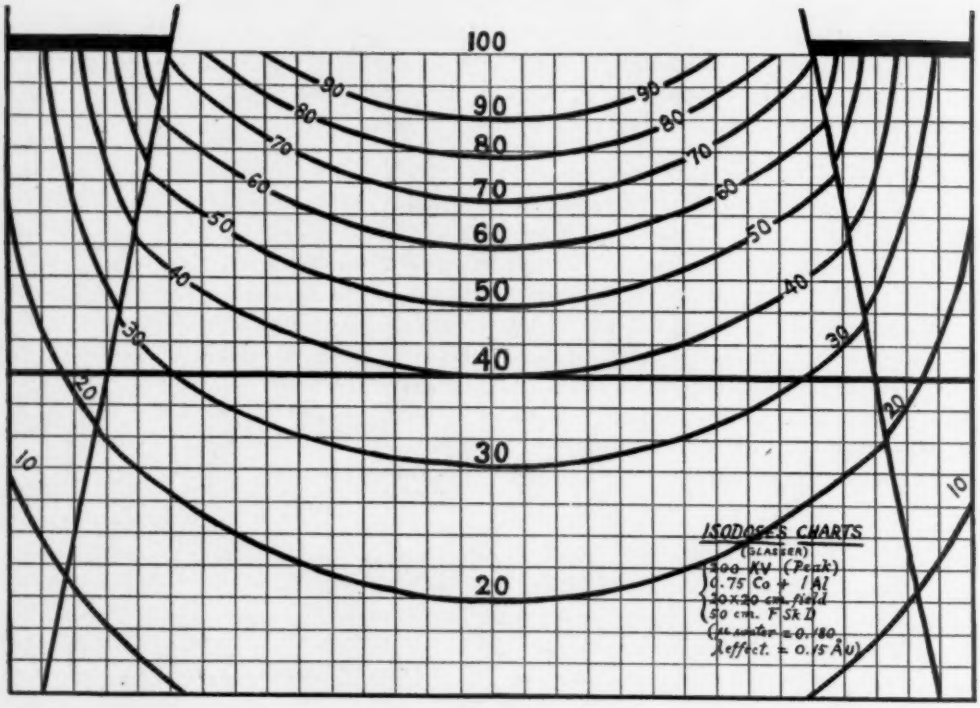

100
90
80
70
60
50
40
30
20
10
ISODOSES CHARTS
(GLASSER)
200 KV (Peak)
0.75 Co + 1 Al
20×20 cm. field
50 cm. F Sk D
(μ water = 0.180
λ effect. = 0.15 Å U)

FIG. 1.

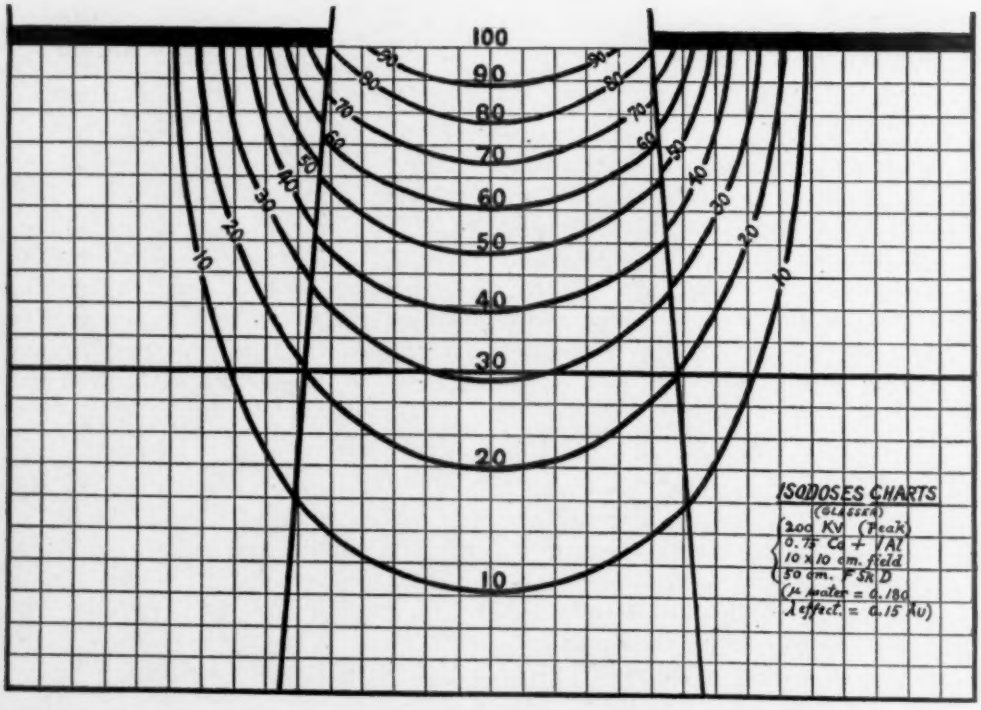

100
90
80
70
60
50
40
30
20
10
ISODOSES CHARTS
(GLASSER)
200 KV (Peak)
0.75 Co + 1 Al
10×10 cm. field
50 cm. F Sk D
(μ water = 0.180
λ effect. = 0.15 Å U)

FIG. 2.

beam, large differences in intensities of the central and edge beams at the same depth, high intensities at the side of the direct bundle below the lead screen, etc.

These isodose charts are intended to supplement the charts and tables now in use.

BIBLIOGRAPHY

1. Kroenig, B., and Friedrich, W. Berlin, 1918.
2. Dessauer, F., and Vierheller, F. *Strablentherapie*, 1921, xii.
3. Duane, W. Am. J. Roentgenol., 1922, ix, 12.
4. Bachem, A. *J. Radiol.*, 1922, iii, 11.
5. Glasser, O. Diss. Freiburg, 1919.

# THE IMMEDIATE EFFECT OF RADIUM AND X-RAYS ON ENZYME ACTION

BY S. CLEMENT ROTH, AND JOHN J. MORTON, M.D.

Section of Surgery, Yale University School of Medicine

NEW HAVEN, CONNECTICUT

THE histological changes which follow radiation of tissues are definite and well known, but the fundamental nature of the processes involved is still to be explained.

Many theories have been advocated[1] and rejected for lack of experimental evidence. The enzyme theory, however, is one of the most recent to be offered, and sufficient data has not been accumulated as yet to test its validity.

Packard,[2] who is the sponsor for the theory, believes that the action consists in the "activating of autolytic enzymes which bring about a degeneration of the complex proteids, etc." The work of Richards[3] is quoted as showing experimental proof in favor of the theory. He used commercial pepsin and the Mett method. After exposure of the pepsin solution to *x*-rays for a short (4 min.), moderate (10 min.), and long time (30 min.), he interpreted his results as acceleration, no effect, and inhibition of the peptic activity for the above factors. He obtained similar results with taka-diastase and starch, using the Benedict method. Willcock[4] claimed to have shown that pepsin, trypsin, and ptyalin were injured by the radium radiations. Brown[5] also thought he showed marked inhibition of the activity of pepsin, and pancreatic diastase after exposure to Radium D, E, and F, but the autolytic ferment of the dog's liver was not affected. Neuberg,[6] on the other hand, found the autolytic ferments to be accelerated; and Bergell and Bickel[7] claimed that the activity of the pepsin was enhanced. Richter and Gerhartz[8] interpreted their results with pepsin exposed to *x*-ray, using nitrogen determinations as the index, as negative; and still more recently, Lawrence,[9] on exposure of physiological diastase to unfiltered radiation from a Coolidge tube (9 in. from the anticathode, 5½ in. spark-gap at 2 ma. in secondary) reported negative findings for the effect on this enzyme.

In view of the conflicting evidence presented above, an attempt was made during the past year to study the effect of radiation on solutions of pepsin.

A review of the literature shows that peptic power is determined by measuring the transformations of various complex substrates by a large number of methods.[10] The dissolving power, the various modifications of the peptonization of the substrate, the use of new substrates, the reversal of procedures, and the application of the increasing number of biochemical methods to the analysis of the products of hydrolytic cleavage—all of these form the basis for methods. Since no method is universally accepted, the following methods were selected for trial: edestin, pea globulin,* and Mett.

Only the immediate effects were observed. The study of the late effects, though desirable, is complicated by the possibility of infection of the solutions. Large amounts of antiseptics, which must be used if the solutions are to be kept sterile for a long time, inhibit and destroy the enzyme. Sterile glassware was used throughout the experiments.

From scale pepsin (Sharp and Dohme), 1 to 3,000, 1 per cent solutions containing 0.22 per cent HCl were prepared.

* Professor Lafayette B. Mendell kindly supplied us with the original pea globulin prepared in his laboratory by Rose.

With edestin[11] and pea globulin[12] as substrates, pepsin solutions were exposed to the gamma rays and to *x*-rays.

Radium, amount, 50 mgm.; filtration, 0.5 mm. silver, 2.0 mm. lead, 1 mm. glass. Distance, none. Time in hours: ¼, ½, ¾, 1½, 4 and 10⅚ respectively.

*X*-ray: Coolidge tube, 6 in. spark (measured between points); 5 ma.; filters, none; 8 in. distance from anticathode to center of solution. Time in minutes: 1/20, 3½, and 7, respectively.

The results showed no differences between the peptic power of the radiated and the control solutions.

With the method of Mett,[13] the solutions were exposed in two series to the gamma rays of radium:

*Series I.* Radium, amount, 50 mgm.; filtration: 0.5 mm. silver, 2 mm. lead, 1 mm. glass. Distance, none. Time in hours: ½ and 16⅚.

*Series II.* Radium, amount, 100 mgm.; filtration, 0.5 mm. silver, 2 mm. lead, 1 mm. glass. Distance, none. Time in hours: ⅔, 4½ and 12⅓, respectively.

The results of the Mett method measured in the number of millimeters of egg albumin digested are recorded below. Each figure represents the average of at least 4 Mett tubes.

| Series | Control | Short radiation | Moderate radiation | Long radiation |
|---|---|---|---|---|
| I | 3.91 | 4.05 | | 4.20 |
| II | 4.00 | 4.00 | 4.03 | 4.02 |

To obtain greater accuracy, gravimetric methods were tried. The weight of the Mett tube before and after digestion, and the difference in these weights was recorded. The error resulting from the presence of digesting fluid at the ends of the tubes was found to be minimized by draining the fluid with a piece of tightly-twisted absorbent cotton. Upon comparison, however, no greater accuracy was gained by this time-consuming procedure.

Human gastric juice, representing more closely a physiological solution, was then exposed to *x*-rays in the following dosage: Coolidge tube, 6 in. spark-gap (measured between points); 5 ma.; 8 in. distance; no filters; time in seconds and minutes: 10″, 3′30″, 7′12″, respectively. This dosage corresponds to ⅛, 3 and 6 MacKee units. The possibility of infection, under the conditions of obtaining gastric juice, could not be excluded. The digestive power of a solution of 15 c.c. of filtered gastric juice diluted to 100 c.c. (0.22 per cent HCl) is shown below. The figures represent the number of millimeters of egg albumin digested, the average of at least 4 Mett tubes.

| | | |
|---|---|---|
| Control solutions | ..... | 1.75 |
| 10″ | radiation ..... | 1.73 |
| 3′30″ | radiation ..... | 1.78 |
| 7′12″ | radiation ..... | 1.78 |

These readings show such slight variations from the control that they are considered to fall within the range of experimental error of the method employed. Close scrutiny of the positive results reported in the literature shows that the effects interpreted may be explained in a similar manner.

## CONCLUSION

From a consideration of the results which follow the radiation of pepsin solutions by *x*-rays and the gamma rays of radium, there does not appear to be any definite effect on the enzyme activity which was determined by the edestin, globulin and Mett methods. Such slight variations as were noted fall within the limits of experimental error.

## BIBLIOGRAPHY


1. MacKee, G. M. *X*-rays and Radium in the Treatment of Diseases of the Skin. New York, 1921, pp. 205–206.
   Simpson, F. E. Radium Therapy. St. Louis, 1922, pp. 86–87, 97–98.
2. Packard, C. The effects of radium emanation on the rate of cell division. *J. Exper. Zool.*, 1916, xxi, 199.
3. Richards, A. Effects of *x*-rays on the action of certain enzymes. *Am. J. Physiol.*, 1914, xxxv, 224.
4. Willcock, E. G. *J. Physiol.*, 1906, xxiii, 207.
5. Brown, T. R. *Arch. Int. Med.*, 1912, x, 405.
6. Neuberg, A. *Verhandl. d. deutsch. path. Gesellsch.*, 1904, vii, 157.
7. Bergell, P., and Bickel, A. *Verhandl. d. Cong. f. innere Med.*, 1905, xx, 157.
8. Richter and Gerhartz. Ueber die Einwirkung der Rontgenstrahlen auf Fermente. *Berl. klin. Wchnschr.*, 1908, xlv, 646.
9. Lawrence. The effects of *x*-rays on enzyme action. *Arch. Radiol. & Electrotb.*, 1922, xxvi, 244.
10. Euler, Hans. General Chemistry of Enzymes. New York, 1912, p. 295.
    Effront, Jean. Biochemical Catalysts in Life and Industry, tr. by Prescott. New York, 1917, p. 249.
    Waldschmidt. Ueber die Verschiedenen Methoden, Pepsin u. Trypsin quantitativen zu bestimmen, nebst Beschreibung einer einfachener derartigen Methode. *Arch. f. d. ges. Physiol.*, 1912, cxliii, 189.
11. Edestin method: See Effront, reference 10, p. 253.
12. Rose. *Arch. Int. Med.*, 1910, v, 469.
    Givens. *Bull. Hyg. Lab.*, U. S. P. H. S., 1915, ci.
13. Mett. Beitrage zur Physiologie der Absonderungen. Weitere Mitteilungen zur Innervation der Bauchspeichedruse. *Arch. f. Anat.*, 1894, xviii, 58.
    Christiansen. *Biochem. Ztschr.*, 1912, xlvi, 257.

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

*Editor:* H. M. IMBODEN, M.D.

*Associate Editors:* JAMES T. CASE, M.D. · H. K. PANCOAST, M.D.

*Publisher:* PAUL B. HOEBER, NEW YORK

*Issued Monthly. Subscription, $6.00 per year. Advertising rates submitted on application. Editorial office, 480 Park Avenue, New York. Office of publication, 67-69 East 59th Street, New York.*

*Information of interest to all readers and lists of officers of The American Roentgen Ray Society and The American Radium Society will be found on the two pages preceding Table of Contents.*

## CHICAGO AGAIN

According to the old patriarchal custom, every seventh year was a jubilee year. The American Roentgen Ray Society met in Chicago seven years ago, and this year we come back there for a great rally meeting.

Last year we went to the Pacific Coast from a sense of duty and to demonstrate that the organization is 100 per cent American. This year we will rally in the great central metropolis for the banner meeting of our history.

The Society has practically doubled its membership since the 1916 Chicago meeting, even with its very conservative plan of development, and its meeting this year will attract a large attendance of clinicians and surgeons who are not primarily roentgenologists.

The meeting will again be in the Congress Hotel, which is centrally located on the lake front and admirably suited for the accommodation of all departments of the convention.

Dr. Hollis Potter, the President-Elect, is Chairman of the Program Committee and already has the program well under way. It is understood that his plan will be to have fewer papers and more detailed discussions, and that he will adhere to the constitutional provision that all papers must be in his hands sixty days before the meeting.

The date of the meeting will be Sept. 18th to 21st inclusive.

## CORRESPONDENCE

*To The Editor:*

Having secured the endorsement of the Council of the American Roentgen Ray Society, I would beg leave to submit through the JOURNAL the following plan for collective investigation by the members of the American Roentgen Ray Society and by other roentgenologists who may be interested in this project.

It seems highly desirable that there be collected for the purposes of study and investigation a large number of films of carefully studied cases. In the inception of this plan there must necessarily be taken up at first only a limited number of subjects, which number could be increased as the details of the plan are worked out.

It is suggested that roentgenologists send in to the below-mentioned address duplicate films of carefully studied cases; these films to be the property of the American Roentgen Ray Society and to be collected for the purpose of cooperative study. It is very desirable that these films should possess a high standard of technical excellence. They should be accompanied by as full clinical, operative, and pathologic data as can be secured. Each film should be carefully labeled so that it can be identified with the clinical history accompanying it.

The subject which has been chosen for the beginning of this collective investigation is "Bone Tumors." It is suggested that at first the films illustrate bone tumors

of the long bones and extremities; also that, as far as possible, cases be selected which admit of a complete study. It is desired that these films should be of sufficient size to include the entire pathologic area and enough of the adjacent healthy area so that in their inspection they can be regarded as inclusive. The films should display the pathology from enough angles to make the study as precise as possible. It is planned that this collection, when classified, shall be demonstrated at the scientific exhibit of the American Roentgen Ray Society at future meetings. A plan will be devised by which lantern slides of typical conclusive cases can be available for the members of the Society.

The second subject which has been selected is "Legg's Disease" (Perthes' Disease). Films illustrating this condition should, if possible, be stereoscopic and must be accompanied by as complete a clinical record as possible. Cases should be selected preferably where the contributor considers that he will have the opportunity for observation of the case over a considerable length of time.

The third subject which has been selected is "Fractures of the Skull in Children." It is desired to secure consecutive films covering the roentgen history of fractured skulls in children. Here also it is desirable that cases be selected which will probably be under observation for some considerable length of time.

The fourth subject selected is "Joint Syphilis." Here it is highly desirable that the clinical and therapeutic data be as accurate and conclusive as possible.

As material is sent in it will be acknowledged in subsequent numbers of the JOURNAL so that the members may be kept informed as to the growth of the collection. Correspondence and suggestions as to the method of handling these studies and the choice of future subjects for study will be very gratefully received.

Directions. Films should be packed in a flat package (*not rolled*) and should be directed to the Department of Roentgenology, University Hospital, Ann Arbor, Michigan, attention of Dr. Hickey. It is earnestly hoped that the roentgenologists of North America will respond to this opportunity of making a collection which will be of the very greatest scientific value.

P. M. Hickey.

March 20, 1893.

*To The Editor:*

It has frequently been noticed that certain dental films after development show a fogging at one end that has the appearance of a small tuft of fine hair, and it was supposed to be due to some light leakage in the film package. However, it was observed in separating the two films preparatory to development that occasionally a static spark was formed, and that when this occurred the films showed the hair-like appearance at one end. This also can be produced experimentally with unexposed films, by pulling them apart repeatedly in the dark room.

The knowledge of this observation has value in careful dark-room technique, as this sparking may easily be avoided by care in separating the films.

Very truly yours,
V. M. Moore.

Jan. 18, 1923.

## THE LEONARD PRIZE

The American Roentgen Ray Society is again offering the Leonard Prize in 1923, details for which appear on advertising page xiii of this number of the JOURNAL. The manuscripts submitted for the 1921 prize were of a high order of merit and covered a variety of subjects pertinent to roentgenology. It is to be hoped that the contestants for the next prize will be equally zealous in their efforts.

*Subscribers to* THE AMERICAN JOURNAL OF ROENTGENOLOGY *visiting New York City, are invited to make the office of* THE JOURNAL (*69 East 59th Street, New York*) *their headquarters. Mail, packages or baggage may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; in this case, kindly notify us in detail as to requirements and prices. List of operations in New York hospitals on file in our office daily.*

# TRANSLATIONS & ABSTRACTS

DALAND, ERNEST M. Radium treatment of keloids. *Surg., Gynec. & Obst.*, Jan., 1923, xxxvi, No. 1, p. 63.

This paper covers a large series of cases treated in the Collis P. Huntington Memorial Hospital, Boston. A small bibliography accompanies the article. As the result of his observations, the author concludes that every keloid can be destroyed by radium if a sufficient dose is used. Silver filtration (1 mm.) should be used in keloids of recent origin, in children, in people of dark complexion and in exposed areas, such as the face. The dosage should be from 30 to 60 mc. hours, according to the age of the patient. Practically unfiltered tubes should be used on all other keloids. The dosage should be from 15 to 30 mc. hours per tube. It should be explained to patients that ulceration will result from this type of treatment. There is no evidence to show that the destructive doses damage the tissues so that the lesions recur. There no lessening of the tendency of an individual to develop keloids.

MOORE, BEVERIDGE H. Subastragaloid dislocation of the foot. *Surg., Gynec. & Obst.*, Dec., 1922, xxxv, No. 6, p. 788.

Subastragaloid dislocation of the foot is a rare injury, alternative with abduction and adduction fractures in the region of the ankle. The physical signs of this form of dislocation are eversion and abduction of the foot, with prominence of the head of the astragalus in the inner side of the foot. In old cases there is lowering of the malleoli and thickening of the foot below the malleoli. Treatment in recent cases is reduction, either open or closed, as may be indicated. Total astragalectomy gives good results in old cases. Excellent roentgenograms and photographs illustrate 3 cases reported in this article.

COHN, ISODORE. Forward dislocation of both bones of the forearm at the elbow. *Surg., Gynec. & Obst.*, Dec., 1922, xxxv, No. 6, p. 766.

Anterior dislocations of both bones of the forearm are rare. Only 23 cases, including the case reported by the author, have been found in a search of the literature. In all cases which have been verified by operation or autopsy, there has been an extensive laceration of the ligaments about the joint and a stripping up of the muscles in the immediate vicinity from the respective bones.

Anterior dislocations of the elbow may be either uncomplicated, or associated with a fracture of the olecranon or coronoid process. A review of the literature suggests that some of the cases have been reduced with very little difficulty.

An excellent bibliography accompanies the article, which deals *in extenso* with the diagnosis and treatment of this condition. The treatment should always be surgical.

MOFFAT, BARCLAY W. Isolated disease of the scaphoid. *J. Am. M. Assn.*, 1923, lxxx, 87.

The author has found 42 cases of isolated disease of the scaphoid since Köhler described the first case in 1908. By roentgen ray the scaphoid is seen as a disc, from one-third to one-fourth normal in size, biconcave, or of irregular density, or opaque, the picture, of course, varying with the stage of the disease. Four new cases are added to the literature.

WHEELON, HOMER. Duodenal motility. *J. Am. M. Assn.*, Mar. 3, 1923, lxxx, No. 9, p. 615.

In view of the demonstration by recent experimenters that duodenal contents are normally regurgitated into the stomach toward the close of gastric digestion, and in view of the frequency with which reverse movements of barium are seen during radiographic examination of the gastrointestinal tract, the author undertook a number of *x*-ray observations following the direct injection of barium into the human duodenum. Barium in quantities of from 20 to 60 c.c. was injected directly into the duodenum without causing distress, the stomach and duodenum having previously been emptied of their contents. The first observed movement of the barium following injection into the duodenum is a more or less complete division of the mass at the point of greatest distention. Following the primary partial division, barium is usually passed along both directions of the duodenum, the central portion being delivered to the upper duodenum or cap, the distal portion to the jejunum. Reverse movements of barium sooner or later result in the complete filling of the cap, at which point barium may rest for a long period of time. Following distention of the cap by reverse movements, barium is passed forward by progressive movements which, at times, carry material through the point of original duodenal distention to lower segments. Barium may be delivered from the point of distention to the stomach as the result of reverse movements. In the majority of instances, barium is passed into the stomach only after several injections of the duodenum. Rhythmic segmental and pendular movements of barium occur in the duodenum. Barium tends to rest at the point of injection—inferior flexure. This

region is the usual point of injection. In one instance of jejunum injection, marked reverse movements resulted in the lodgment of barium in the cap, stomach and inferior flexure.

HOLLANDER, EDWARD. Fallibility of roentgenologic evidence of healed gastric ulcer. *J. Am. M. Assn.*, Jan. 6, 1923, lxxx, No. 1, p. 29.

Roentgenologic examination affords the best method of obtaining definite objective record of morphologic changes in the surface of an ulcer. It seems reasonable to interpret the diminution in size or the total disappearance of a niche as evidence of healing or cure, especially when these changes are accompanied by clinical evidence of improvement, and by other roentgenologic findings, such as the disappearance of an incisure, and improved motility. However, the author presents a case which shows that this interpretation is open to error. There are causes for the disappearance of the niche other than the obliteration of an ulcer crater by granulation or cicatricial tissue. Food may enter the crater. In the case presented mucoid material filled the crater. It has been suggested that pressure by edema or enlargement of an organ, such as the liver or pancreas, adjacent to the ulcer, may obliterate the crater. It must further be borne in mind that, even if an ulcer crater is filled with granulation tissue, the ulcer cannot be considered healed unless its surface is completely covered with epithelial tissue. This condition the roentgen ray cannot demonstrate. In the case cited by the author, the ulcer was diagnosed roentgenologically on April 30th; the medical cure announced as proved by the same method on July 2nd, but on August 3rd, at operation, a callous ulcer was found present on the lesser curvature at the point corresponding to the niche in the roentgenogram of April 30th.

SCHROEDER, J. HENRY. Sarcoma (?) of thigh, with secondary sarcoma (?) of inguinal region, liver and lungs: Recovery after deep roentgen irradiation. *J. Am. M. Assn.*, Jan. 6, 1923, lxxx, No. 1, p. 23.

The author cites a case of a man, aged fifty, injured by crushing of right thigh. Roentgenograms do not reveal any injury to the bone. After six months a clinical diagnosis of sarcoma of the thigh was made, and amputation advised. Routine examination with the *x*-rays revealed extensive and typical secondary sarcoma of both lungs. From Jan. 21st to Feb. 1st, the thigh, inguinal region and both lungs were irradiated, each region receiving in its depth the full dose in one sitting (for the lungs, 70 per cent of erythema dose at 200 kv., and for the other areas, 80 per cent of the surface erythema dose at 220 kv.). The sarcoma dose was again administered to the thigh and the lungs between March 22nd and March 27th. At the beginning of the third period of irradiation, June 29th, the liver was found to be markedly enlarged and very tender, and at this time the liver and the thigh were each given the sarcoma dose. During the fourth period of treatment, August 21st, the thigh received its last irradiation. This excessive irradiation was well tolerated. There were no alarming blood changes after the various doses. At the present time the patient is free from any evidence of the disease, his general condition being excellent. Only the muscles of the leg have not gained their former strength. The lungs became entirely clear after the second dose.

The author feels that the case demonstrates that widely disseminated sarcoma is in itself not a bar to successful treatment with deep roentgen irradiations. Nine months ago this patient was lying in a hospital, hopeless and helpless; today he is looking forward to a renewed useful existence.

STOKES, J. H., and GARDNER, BOYD S. The demonstration of unerupted Hutchinson's teeth by the roentgen ray. *J. Am. M. Assn.*, Jan. 6, 1923, lxxx, No. 1, p. 28.

In any large syphilologic practice, cases are occasionally encountered in which the fact that the child's teeth are still of the first dentition makes it impossible, in the absence of other conclusive evidence, to clinch the diagnosis of heredosyphilis by the identification of true Hutchinson's teeth. In such cases, it seems possible that intra-alveolar identification of Hutchinson's teeth by the reontgenogram may be of diagnostic service. It is only necessary to study the unerupted upper incisors to get the full benefit of the dental examination in such patients.

THEDERING. *X*-Ray stimulation doses and diseases of the hair. *München. med. Wchnschr.*, Oct. 6, 1922, lxix, No. 40, p. 1430.

The effect of the epilation dose on the healthy hair demonstrates absolutely the meaning and extent of the Arndt-Schulz biologic basic law: "Weak stimuli increase, moderate stimuli inhibit cellular activity; strong stimuli destroy the cell." In raying the beard with the epilation "11 X" doses through ½ mm. aluminum filter we may observe increasing growth of the beard due to the gradually increasing wave of reaction. Patients voluntarily state that they need a shave more frequently. After a fortnight the beard stops growing and with the third week epilation sets in.

The theoretic object of applying roentgen radiation to the hair is threefold and corresponds to the three different biologic stimulation doses. It aims to stimulate slow-growing hair; temporarily to remove diseased hair; permanently to remove troublesome hairy growth (beard in women).

Temporary epilation is mostly employed in parasitic diseases of the hair follicles—sycosis and trichophytosis—and has been practiced for over a decade in *x*-ray therapy. Permanent epilation in inconvenient bearded growth has likewise become common practice and proved a safe, harmless, successful method if applied with a sufficiently thick (4 mm.) aluminum filter by an experienced specialist.

The *stimulation dose* is little employed, if one is to judge by the published reports. In 1900, Kienboeck, and about that date Holzknecht also, published reports of successful roentgen radiation in alopecia areata. However, their suggestions were forgotten, probably because they applied the epilation dose which first provokes depilation and stimulates the paralyzed papilla only after the reactionary wave has subsided. Total depilation for three months may actually be considered too radical a process on account of a few individual foci in alopecia areata, all the more since Nagelschmidt and Bering successfully apply the *quartz light* and have found it a milder therapeutic agent for insuring a new growth of hair. We may thus well understand why such eminent radiologists as Wetterer and Krohmayer refuse to treat alopecia areata with the *x*-rays.

So long as the technique of dosage was not perfected, it was reasonable to look askance on a method in which the wrong dose might induce temporary or even permanent depilation instead of stimulating the growth of hair. The modern technically educated roentgenotherapist need not fear this criticism.

The author believes himself the first to suggest substituting *x*-ray stimulation dose, based on the above biologic principle, for quartz light treatment in alopecia totalis, in his book on "Quartz Light," 1916, and his article, "Radiotherapy in Alopecia Totalis," 1916. The stimulation dose consists in raying the scalp with about ½ of the epilation dose through ½ to 2 mm. aluminum filter through four fields once in two weeks until the hair grows again. In the course of years the author has increasingly substituted for quartz light treatment this *x*-ray stimulation radiation in alopecia areata, seborrhea, etc. It has the advantage of more pronounced, permanent and deep effect; of absolutely certain detection of hidden foci in systematically raying the thick hair of women. Quartz light therapy of these cases requires careful and time-consuming parting of the hair and even then beginning foci may be easily overlooked.

The excellent results obtained in various diseases of the hair are discussed by the author in his recent publication, "Diseases of the Hair and Baldness: Prevention and Treatment with Light and Roentgen Rays," Oldenburg, 1922. It is intended to prove that roentgen stimulation dose, as the most modern idea of scientific therapy, may be successfully employed in those diseases.

PURCELL, C. E. An Interesting *X*-Ray Study of a Foreign Body: Honey Locust Seed in the Right Bronchus. *Kentucky M. J.*, Aug., 1922, xx, 529.

The author reports a case of a child of twenty-four months who swallowed one of two honey locust seeds. The first *x*-ray plate gave an absolutely negative finding. The companion seed, which the parents brought along, was then placed on an *x*-ray plate and gave a good shadow of the seed. This companion seed was then placed on a plate underneath the child and its position marked by a pair of hemostats. This roentgenogram showed the companion seed very plainly. There were no physical signs of obstruction in the lungs—no difficulty in breathing and no other symptoms to suggest the presence of a foreign body in the lung. Laryngoscopy revealed a severe laryngitis. By noon of the following day it was observed that the child had some difficulty in breathing when it played or walked around the square. It became irritable and developed a slight rise of temperature. Under general anesthesia a bronchoscope was passed and the seed easily removed from the right bronchus.

The author concludes, therefore, that a foreign body can remain in the air passages without any symptoms, especially early symptoms, during the first twelve hours. The published roentgenograms do not reveal any signs in the chest which would indicate pulmonary changes due to the presence of a foreign body.

DAELS, FRANS. Histological Pictures Representing the Cure of Uterine Basocellular Epithelioma. *Proc. Roy. Soc. Med.*, Apr., 1922, xv, 19.

According to the author, in proportion to the intensity of the action of the irradiation upon the cancer cells of the uterine basocellular epithelioma, the following phenomena are observed. They are changes not met with in cases of spontaneous degeneration, namely:

1. Massive and rapid caryorrhexis of the cancer cells without any intervention on the

part of the blood-cells or any profound degeneration of the normal tissues.

2. Progressive necrosis occasioned by pycnosis or achromatosis, with eosinophilia or vacuolization of the protoplasm and phagocytosis by polynuclear blood cells; this destroys quite a large number of alveoli; this well-marked phenomenon is here specially characteristic of the action of the irradiation.

3. The transformation of the cancer cells into giant cells and giant nuclei—an alteration that may lead to necrosis with invasion by polynuclear leukocytes, or to a gradual atrophy with fatty degeneration of the protoplasm and disappearance of those elements without any participation of leukocytes in the process.

The action exercised by the irradiation—radium irradiation more especially, but however not exclusively, since one often added irradiation by *x*-rays after radium treatment—first affects the nucleus and sets up an actual rupture of the nucleus comparable to the action of the irradiation upon the lymphocytes: or else a destruction of the nucleus, sometimes combined with eosinophilia or the megakaryocytic-shaped degeneration which seems to result from nuclear fusions due to loss of karyokinetic power.

The author's observations lead him to admit that the polynuclear leukocytes only occur in association with spontaneous degenerations or with radio-therapeutic transformation of the basocellular epithelioma, as a consequence of incidental infections or the necrosis of cancer cells, and that they do not take an active part in the elective regression proper. And so it must also be admitted that the connective tissue has no active participation in the regression proper, as one meets with the most excellent definition of regression in the midst of the empty space remaining after the disappearance of the cancer cells. On the other hand, it seems that a special significance of effective reaction against the cancer proliferation or its agent must be ascribed to infiltration of leukocytes.

4. The appearance of giant cells without the characteristics of malignant tissue, sometimes with a distinct follicle shape, the appearance of true histological follicles as a consequence of radium irradiation of cancer alveoli, lead us to the adoption of the hypothesis of the liberation of a germ or agent to which the body is supposed to react by lymphocytic infiltration and formation of giant cells. The observation should be connected with the two different sarcomatous and follicular reactions of the body upon the bacillus of Koch. The histological appearances found upon the healing of the follicle that arises after experimental injection of killed Koch bacilli should be remembered in this connection, namely, formation of giant cells, fusions of the nuclei, formation of megakaryocytic elements, progressive liberation and atrophy of these megakaryocytes. These phenomena have the greatest resemblance to those observed in the case of cancer regression.

SMITHIES, FRANK. Necessity for Caution in the Employment of High Voltage Roentgen Rays as a Therapeutic Agent Against Malignant Disease: Acute Adrenal Insufficiency and Death as Sequelae. *Surg., Gynec. & Obst.*, Jan., 1923, xxxvi, No. 1, p. 61.

The author of this paper has evidently failed to note the numerous warnings with which the literature of deep roentgenotherapy is filled, and states that "literature has carried very few, if any, criticisms upon or warnings against the new roentgen therapy." Nevertheless, the details of a case of fatal acute adrenal insufficiency following deep therapy is very instructive. The patient, aged fifty-eight, was treated by deep therapy on an erroneous diagnosis of sarcoma of the spine. The author's report of the amount of treatment given is too vague to warrant repetition here. Within a month following the roentgen therapy the patient began to experience unusual fatigue upon slight exertion, and within two months the diagnosis of bilateral adrenal insufficiency was clear to the author. In spite of intravenous injection of adrenalin and salt solution in heroic doses, followed by partial return to consciousness, the patient died from exhaustion and cardiorenal failure. Unfortunately no post-morten study was permitted.

The author summarizes the evidence in this case as follows: "Instances of 'acute' or 'fulminant' Addison's syndrome are rare; it is most unusual to have the disease appear after the age of fifty and exitus to occur in less than a year. An instance of death from the Addisonian syndrome, within four months after 'deep' roentgen therapy of long duration and very high voltage, in a man aged fifty-eight is recorded. The roentgen seances were given as curative measures for supposed malignancy of the spine (a diagnosis subsequently shown to have been faulty). It can scarcely be presumed that this patient's fall from his horse produced simultaneous acute, double-sided adrenal injury and failure. The man was very 'fit' when he sustained what seemed to be nothing more than muscle bruises to his paraspinal group. He remained 'fit', despite his slight muscular lameness, until a few weeks after roentgen exposures, and his collapse afterward was rapid.

"The proximity of the adrenals to the areas

treated by the high voltage *x*-rays for long time-intervals strongly suggests that this form of therapeusis was an agent responsible for the acute collapse of adrenal function and doubtless the destruction of chromaffin tissue. Unfortunately, autopsy was denied: the cause of the disability and death were, however, unmistakable."

HYMAN, A. Diverticula of the Bladder in Children. *Surg., Gynec. & Obst.*, Jan., 1923, xxxvi, No. 1, p. 27.

Vesical diverticula are rare findings in childhood, not more than 30 being found in a review of over 600 cases.

Diagnosis of this condition in children should present no difficulties. Little value can be placed on the history. Physical examination of the abdomen is of considerable importance. A chronically distended bladder especially asymmetrical in outline, should make one suspicious of a diverticulum. The cystoscope and the cystogram are absolutely essential in making a correct diagnosis. Of the two, the cystogram will be found more valuable. Cystoscopy is at times difficult in the young, especially in the presence of a marked pyuria, when it may be impossible to cleanse the bladder sufficiently to obtain a clear picture. Even cystoscopy gives no idea of the size and shape of the pouch, although it reveals the presence of the orifice. Cystography can be performed, even in very young children, without the slightest difficulty, and requires no anesthetic. The roentgenograms should be taken in different positions, otherwise a small sac may be overlooked or the shadow of the diverticulum may be obscured by that of the bladder itself. In young children conditions are especially favorable for obtaining roentgenograms in the lateral as well as in the anterior positions. Additional information may be obtained if one makes a plain roentgenogram, after which the solution is drained off and air injected into the bladder and another plate taken. The air-filled bladder stands out in contrast to the sacs which still contain the opaque solution.

The presence of diverticula in patients so young, without evidence of any obstruction, leads one to conclude that they were congenital and that in all probability many of the diverticula in adults have also a similar origin.

HOCHSTETTER, F. Duodenal Stenosis Following an Ancient Tuberculous Peritonitis. *Fortschr. a. d. Geb. Röntgenstrahlen*, xxix, 2.

This case is interesting because of the successful roentgenological diagnosis made of duodenal obstruction. The stomach and the duodenum were converted into one large sac without any visible separation at the level of the pylorus, both organs having the caliber of a dilated stomach. Operation revealed a very tight stenosis of the small intestine at the jejunoileal junction and the presence of multiple adhesions involving all the organs of the abdominal cavity.

CHAPPEL, HALBERT W. The Necessity for an Immediate and Thorough Roentgenological Study of All Injuries to the Spine. *Calif. State J. M.*, Dec., 1922, xx, 436.

The author cites 15 cases in which roentgenological examination was of the greatest assistance in connection with injuries to the spine, and in many of which a thorough roentgenological study immediately following the injury would have been the only means of arriving at a correct diagnosis.

KEIFFER. The Mechanism of Retrogression of Human Uterine Fibroids. *J. de Radiol.*, 1922, ix, 95.

According to the author, the mechanism of the diminution in size of uterine fibroids in the human is variable, and he has only been able to establish clinically a diminution in volume and a softening of the fibrous nuclei, and, with the microscope, atrophy with progressive sclerosis. In the course of and following pregnancy, certain uterine fibroids may disappear by a complex mechanism, especially that of a light lipolysis associated with other degenerative processes. After the menopause the retrogression is observed in the form of a simple sclerosis. In hysterectomies performed after the application of radium or *x*-rays, the author has frequently found a veritable central necrosis of the fibrous nuclei associated with all the other signs of degeneration observed in the fibrous uterus after pregnancy and in the course of the physiological menopause.

COOKE, A. B. When Appendicitis Is Not Appendicitis: A Case of Diverticulitis of the Cecum. *J. Am. M. Assn.*, Feb. 25, 1922, lxxviii, 578.

The author reports a case in which an operation for what had been diagnosed as appendicitis revealed a well-marked diverticulitis of the cecum, the appendix not being involved. A total excision of the cecum was successfully performed. The specimen showed a diverticulum of the cecum which had harbored a concretion. This concretion as it increased in size caused a pressure necrosis and an extensive inflammatory deposit. This case is interesting to the roentgenologist as illustrating some of the diagnostic possibilities in the ileocecal region.

SAGET, M. The Intensiometer. *Bull. et mém. Soc. de Radiol. méd. de France.* Nov., 1922, No. 93.

The intensiometer is based upon the same principle as the ordinary iontoquantimeters, but it possesses certain advantages over those without their inconveniences. As its name indicates, it is intended to measure the intensity of ionization, and gives, therefore, the measure of intensity of a given bundle of *x*-rays. This ionization chamber consists of a series of paper discs covered with a thin layer of carbon conductor, but held apart parallel to each other at an interval of 1 cm. by peripheral isolating rings. These discs are alternately connected to the two terminals of the ionization chamber, and constitute thus a sort of condenser with parallel sheets of which the dielectric will be the contained air ionized by the *x*-ray which passes through it.

A double envelope of lead and aluminum constitutes the lateral walls and assures protection against diffusive radiation. The upper surface, through which passes the radiation to be measured, consists of a sliding drawer, permitting easy insertion of the filters to be used. The lower face of the instrument consists of a wooden plate 22 mm. thick, pierced by a groove to receive the ionization chamber of Solomon's ionometer for the purpose of comparing the measurements furnished by the two instruments.

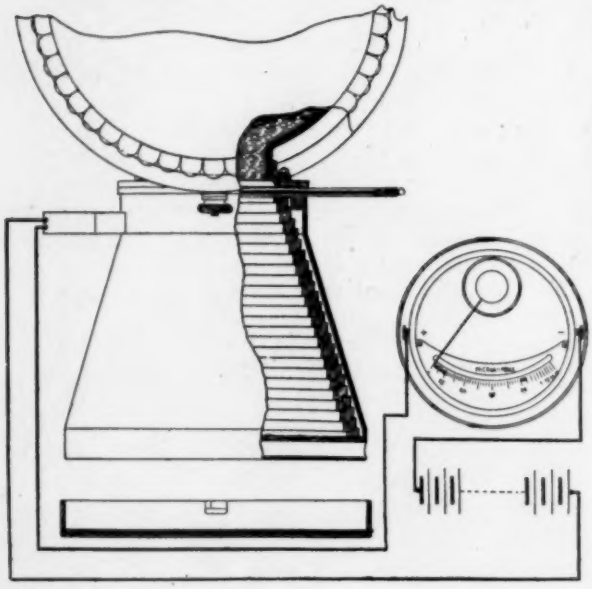


FIG. 1.

The external dimensions of the cone are as follows: Inferior diameter, 29 cm.; total height, 26 cm. It corresponds to a cone of 25 cm. in diameter for a distance of 40 cm. from the tube-target. The volume of the ionization chamber at this distance is about 11 liters. This ionization chamber is placed in the circuit of a storage battery with a direct reading needle galvanometer. This apparatus is as sensitive as can be practically constructed. It registers a deviation as small as two micro-amperes.

FIG. 2.

The storage battery especially designed for this work consists of a hundred Fery dry cells, small model, which should give service during several years without repairs.

The different apparatus are connected with each other by a very supple cable having two conductors perfectly isolated and not permitting any appreciable loss by ionization.

The use of this apparatus is very simple. Under the influence of radiation the different layers of air of the apparatus are ionized and permit the passage of a current, the intensity of which is instantly read on the galvanometer.

This intensity is rigorously proportional to the intensity of the radiation. The readings depend upon the absorptive power of the filters employed.

The intensiometer can be utilized each time it is necessary to determine the thickness and the nature of filters. It is indispensable in case one desires a constant control of the good functioning of his tube or generator. It gives an instant reading of the measure of the intensity of the radiation employed, just as the milliamperemeter gives the measure of current passing through the tube.

The diameter of the orifice through which the ray passes is 25 cm. One can reduce this dimension by the employment of lead diaphragms, the form and dimensions of the opening being determined according to the case.

The ionization chamber is intended to be attached to the tube holder just as the cylinder diaphragm is ordinarily attached to tube holders employed by American roentgenologists. In France this is especially interesting because the holders for maintaining the oil-immersed tubes can be touched by the patient with impunity. Doubtless before long, modifications of this apparatus will be available for use with American instruments.

HERMAN, N. B., and VON GLAHN, WILLIAM C. Carcinoma of the Supra-Ampullary Portion of the Duodenum. *Am. J. M. Sc.*, Jan., 1921, No. 1, clxi; No. 586, p. 111.

One case is reported: Male, seventy-eight years old, complaining some months, upset stomach; lost 10 lbs. in a year; no bloody stools.

*Laboratory Findings.* Blood. Red blood cells, 3,048,000. White blood cells, 5,750. Hemoglobin (Sahli) 45 per cent. Differential formula showed a slight polymorphonuclear leukocytosis.

Urine. Sp. gr., 1.010 to 1.015. Persistent trace of albumin. Granular casts.

Sputum. Positive for tubercle bacilli.

Stool. Bile present. No ova or parasites. Positive for occult blood. (Benzidine and guaiac tests.)

Test-meal. 100 c.c. of brownish fluid. Bile, 0. Lactic acid, 0. Test for occult blood strongly positive. (Benzidine and guaiac tests.) No Oppler-Boas bacilli. Free HCl, 17 per cent. Combined, 16. Total, 33.

Wassermann. Negative.

*Roentgen-Ray Report* (Dr. J. W. Pierson). Chest. Marked spotty infiltration, both uppers, more extensive on the left than on the right; tuberculous in origin.

Gastrointestinal Series. Stomach lies low in the pelvis: marked filling defect in region of antrum pylori, probably due to pressure from lumbar spine; pylorus intact; no retention; duodenal cap well filled. Due to weakness, the patient could not be fluoroscoped.

*Course in Hospital.* Patient became progressively weaker. He vomited small amounts of fluid material at times. His chief complaint was difficulty in swallowing, but since a stomach-tube passed easily this was thought to be due to a reflex cardiospasm rather than to an organic esophageal obstruction. On the night of February 10th he showed active delirium. On February 11th he sank into a semicomatose state and died February 12th.

*Clinical Impression.* Ulcerative pulmonary tuberculosis. Carcinoma of the stomach. Secondary anemia.

*Autopsy, No. 1592. Anatomic Diagnosis.* Chronic fibroid pulmonary tuberculosis with bronchiectasis; ulcerative tuberculous enterocolitis; conglomerate mass of tubercles in the wall of the aorta; generalized miliary tuberculosis; adenocarcinoma of the duodenum; thrombosis of the right common iliac and right pulmonary arteries, left common iliac vein and prostatic veins; arteriosclerosis; right inguinal hernia, undescended testicle, right; brown atrophy of the heart.

Stomach and Duodenum. The stomach is moderately distended with gas and fluid, especially the pyloric portion. The duodenum, just beyond the pylorus, is enlarged and a dense mass can be felt in it, apparently entirely filling the lumen. The serous surfaces of the stomach and duodenum are smooth and glistening.

When opened, the stomach shows no changes. A finger can be readily introduced through the pyloric orifice, but the opening is eccentrically placed and a firm mass can be palpated in the duodenum, completely encircling the finger. This mass is a large, oblong, solid, polypoid tumor, attached to the inferior wall of the bowel and arising just beyond the pylorus. It measures $6 \times 5 \times 3$ cm. in its greatest diameters. The base is somewhat smaller than this. The surface of the tumor is smooth and rounded, with small hemorrhages here and there. The upper surface is hollowed out into a deep gutter with steep sides, which gradually taper as they extend upward, and, conforming to the curve of the bowel wall, they overlap above, forming a tunnel through which the gastric content passed. It was into this tunnel that the finger readily entered. When the bowel was opened these margins sprang apart. The tumor is grayish white in color and cuts with some resistance. The cut surface is rather homogenous in appearance and is dotted with a few yellow flecks. The growth seems to

penetrate only a little way into the wall of the bowel. The duodenal mucosa is gray and translucent and is reflected for a short distance upon the base of the tumor. The folds of the mucosa are well defined and prominent. The ampulla of Vater is negative. The regional lymph-glands are not enlarged and there are no metastases visible in the liver.

*Microscopic Examination.* The tumor is composed of columnar epithelial cells, having a definite glandular arrangement. Often these cells are heaped up into several layers. There is an abundant and rather dense connective-tissue stroma in which are many capillaries and which is infiltrated with small round cells, plasma cells and leukocytes. The gland-like spaces enclosed by the epithelial cells are frequently filled with leukocytes. Small areas of necrosis are seen. At the base the rather orderly adenomatous arrangement is lost, and the cells are more irregular in size and shape and grow in solid cords, penetrating into the circular layer of muscle. Mitoses are fairly numerous. The mucosa can be traced upward upon the tumor for a short distance, then it ends abruptly. In appearance and arrangement the tumor cells resemble more closely those of the glands of the mucosa than those of Brunner's glands. In none of the sections of the regional lymph-glands could any metastases be found. Sections of the liver and pancreas were likewise negative for metastases.

SANTE, L. R. Pneumoperitoneum as an Aid in the Diagnosis of Subdiaphragmatic Conditions. *J. Am. M. Assn.*, Feb. 17, 1923, lxxx, No. 7, p. 464.

The author has found pneumoperitoneum of so much value in examinations of acute infectious involvement of the subphrenic space that it seems advisable to call attention to this special phase of pneumoperitoneum work. Very little air is necessary in the examination of this region, and, if inflation is carried on with the patient in a recumbent position under the fluoroscope, any involvement of this space can be at once detected, and the inflation discontinued. If the procedure is carried out in this fashion, there need be little fear of breaking any existing adhesion walling off an infectious process, no matter how delicate or recently formed. Information gained in this manner, either establishing the presence of a subdiaphragmatic abscess or excluding its possibility, is of the utmost value in the prognosis and treatment of the condition. This is especially true since the accepted method of surgical procedure for drainage of such abscesses is very extensive and necessarily involves vital structures. The author cites a number of cases where this method assisted materially in the difficult diagnosis including empyema, perinephritic abscess, cardiospasm, adhesions of the viscera to the diaphragm, and hernia of the hollow viscera through the diaphragm.

COOPER, J. R. Fibroma of Stomach. *J. Am. M. Assn.*, Feb. 24, 1923, lxxx, No. 8, p. 549.

The author reports a case of a male, aged forty-five, suffering from gastric and intestinal hemorrhages. Two years previously the patient at times became dizzy, and his stools after these spells were dark and of a tarry consistency. A year later, his dizzy spells became more pronounced and he occasionally vomited blood in considerable quantities. The pre-roentgen diagnosis was ulcer of the stomach or duodenum. The roentgen study before administering the opaque meal showed, extending into the gas bubble of the stomach, a tumor-like mass about as large as a medium-sized orange. It was observed to rise and fall with respiration, and no pulsation could be seen. With the stomach filled with the barium mixture the tumor could not be seen. Aneurism of the abdominal aorta was thought of; but the Wassermann reaction was negative and there were no pulsations. At operation a fibromyoma, the size of an apple, was removed from the cardiac end of the stomach. The tumor was growing from the anterior wall, about an inch and a half from the cardia. The pathological report showed fibromyoma with a large ulcer in the mucous surface.

KNAPP, HARRY B. Dislocation of the Semilunar Carpal Bone. *J. Am. M. Assn.*, Dec. 9, 1922, lxxix, 1992.

The mechanism of semilunar carpal bone dislocation is the same as that which produces the common Colles' fracture, this mechanism being the sudden, forcible hyperextension of the wrist. If the anterior radiocarpal ligament is ruptured by the hyperextension, the semilunar carpal bone is apt to be dislocated anteriorly. This ligament usually does not rupture, but the radius, instead, is fractured. Thus fracture is many hundred times more common than dislocation.

Diagnosis is easily made from a roentgenogram taken in the lateral position, which shows the moon-shaped bone more or less completely rotated forward, boldly jutting out of its place in the general alignment of the wrist structures. The length of the hand appears shortened, owing to the settling of the os magnum into the space left by the semilunar. The anteroposterior diameter of the wrist is increased and the semilunar bone may be felt as a hard, perhaps movable prominence beneath the flexure tendon in the anterior wrist region.